INFANTILE PARALYSIS

IN

MASSACHUSETTS

DURING

1910,

TOGETHER WITH REPORTS OF SPECIAL INVESTIGATIONS MADE IN 1911 BEARING UPON THE ETIOLOGY OF THE DISEASE AND THE METHOD OF ITS TRANSMISSION.

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THE STATE BOARD OF PUBLICATION.

THE OCCURRENCE OF INFANTILE PARALYSIS IN MASSACHUSETTS IN 1910.[1]

Reported for the Massachusetts State Board of Health by Robert W. Lovett, M.D., Boston, and Philip A. E. Sheppard, M.D., Boston.

The last year has been one of considerable progress in our knowledge of infantile paralysis. The disease has been widespread in the United States; 23 States have made the disease reportable, and several State boards of health have taken up the work of investigation. The clinical study of cases has become more accurate and careful, and the importance and frequency of abortive cases has been much more fully recognized.

Three certain interesting contributions to our knowledge are as follows: —

Osgood and Lucas found an active virus in the nasopharyngeal membrane of one monkey, who had recovered from the acute attack, five and one-half months afterward, and in another six weeks afterward. This virus was sufficiently active to cause paralysis in a first and second generation of monkeys into which it was injected.[2]

The second contribution is from Flexner and Clark.[3] It has been previously shown that if hexamethylenamin is administered by mouth, its presence can be detected in the cerebro-spinal fluid. Flexner and Clark showed that if the drug be given by mouth, and its use continued after its presence in the spinal fluid can be detected in monkeys, and if such animals are subsequently inoculated either subdurally or by the intracerebral route, the paralysis is in a certain proportion of cases delayed or prevented. But successful results by the drug have so far been obtained in inhibiting infection, and not in restraining an already established infection. In other words, there is as yet an absence of experimental evidence to show that the drug is of much use when the disease has begun.

A third contribution by Anderson and Frost[4] is as follows: It is only very recently that it has been found possible by a laboratory method to

[1] Reprinted from the Boston Med. and Surg. Jour. May 25, 1911.
[2] Jour. Am. Med. Asso., Feb. 18, 1911, p. 495.
[3] *Ibid.*, Feb. 25, 1911, p. 585.
[4] *Ibid.*, March 4, 1911, p. 663.

throw back light on whether a suspicious case occurring in the past may or may not have been abortive infantile paralysis. It is possible by means of neutralization tests to determine this matter by mixing the blood serum of the recovered case with the filtered virus, and injecting the mixture into a monkey, the serum of recovered cases having the power of neutralizing a certain amount of the virus.

By a study of the changes in the cerebro-spinal fluid in monkeys preceding the paralysis, a forecast of the probable changes in man was worked out, and paralysis predicted in a human case where a lumbar puncture was done early. The case is reported by Frissell.[1] The changes are the following: At the height of the lesions in the meninges it has been found in monkeys and confirmed in a human case that the cerebro-spinal fluid is slightly turbid or opalescent, and contains an excess of protein. There is a large increase of white cells, both poly- and mononuclear. The fluid may in some cases be distinctly turbid, and polynuclear cells may predominate. Paralysis occurs at the height of these changes, and then the changes described in the fluid begin to disappear.

In the matter of etiology more attention has been paid to dust, and the data reported by Hill [2] are suggestive. In Winona, Minn., there were 29 cases up to the end of July, 27 occurring on dusty streets. On August 5 street watering was begun at the suggestion of the State and local health officials, and no case occurred in Winona after August 12, although the greatest incidence throughout the State had yet to develop, and the neighborhood outside of Winona continued to show cases. Hill quotes also three other instances in the State of a similar occurrence.

The need of quarantine is being appreciated, and the experience in Nebraska is worth quoting:[3] "We are informed that no legal provision had been made for quarantining such cases, and isolation was recommended, but, of course, could not be enforced, nor could the impending Fourth of July celebration be abandoned. This celebration was well attended, and shortly after cases were reported from communities previously free from the disease, and having had representatives present at the celebration. From May 30 to July 4 we had in the Stromsberg territory 30 cases, while from July 4 to August 3, just thirty days, we had 115 cases." A quarantine was declared necessary, and put into effect on July 22, and after August 3 there were only 15 cases in the Stromsberg territory.

The salient points in progress may be summarized as follows: —

(1) A decidedly keener attitude on the part of the profession, the health boards and legislative bodies toward the disease.

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[1] Jour. Am. Med. Asso., March 4, 1911, p. 661.
[2] Trans. Prev. Med. of Am. Med. Asso., 1910.
[3] Anderson: Pediatrics, August, 1910.

(2) The knowledge that the infection may persist in the nasopharynx in monkeys for long periods.

(3) The possibility of diagnosticating after recovery the character of abortive cases.

(4) The possibility of early diagnosis by means of lumbar puncture.

(5) The possibility that hexamethylenamin may be in monkeys at times a preventive of infection, but does not apparently control established infection.

During the year 1910 the Massachusetts State Board of Health continued for the fourth consecutive year the investigation of infantile paralysis as it occurred in the State.[1]

The disease having been made a reportable one in November, 1909, the Board received notice of cases reported by physicians, and to them blanks were sent to be filled out and returned. Dr. Philip A. E. Sheppard and Dr. T. P. Hennelly were employed by the Board to investigate the cases at their homes, to make an intensive study of epidemic centers and to see as many other cases reported as practicable. The scope and direction of the inquiry have again been supervised by an advisory committee, consisting of Dr. Theobald Smith, Dr. Milton J. Rosenau, Dr. James H. Wright and Dr. John Lovett Morse, who have met at frequent intervals the secretary of the Board and the members concerned in the inquiry. The Legislature of 1910 generously appropriated the sum of $5,000 for the purposes of this investigation.

The data relating to the disease in the State during the year 1910 will be here presented, and later, in another form, will be presented those conclusions that seem warranted by a study of the disease in Massachusetts for four years, with certain general considerations with regard to the disease. The investigation will be continued in 1911 on a more extended scale, $10,000 having been appropriated for the purpose by this year's Legislature.

There are three classes of cases to be reported on: —

(1) The total cases reported by physicians with their location, — 845.

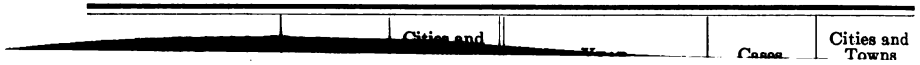
(2) The number of cases for which blanks were filled out, — 601.

(3) A group of cases carefully studied by the medical investigators of the Board, — 200.

The groups of cases considered in various respects will, therefore, differ in number, more space in general being allotted to the 200 carefully studied than to the others.

The record of the last four years is as follows: —

[1] Boston Med. and Surg. Jour., 1908, CLIX, p. 131; *Ibid.*, 1909, July 22; *Ibid.*, 1910, CLXIII, p. 37; Am. Jour. Pub. Hyg., November, 1910, p. 875.



An investigation of the nearness of the affected house to railroad and highroad showed no preponderating number close to either, 15 per cent.

being close to a railroad and about 60 per cent. on the highroad. The nearness to water in the 200 cases studied in and around Springfield

Digging or *excavating* in the neighborhood of affected houses had been in progress in a little over 15 per cent. of 334 cases where data existed on this point.

Presence of Insects, Vermin, Rodents, etc.

Among 185 families, 185 had insects, etc., as follows: —

	Families.
Flies were present in	185
Mosquitoes were present in	67
Ants were present in	62
Mice (house) were present in	60
Bedbugs were present in	39
Roaches were present in	29
Spiders were present in	26
Rats were present in	23
Biting flies were present in	7
Field mice were present in	4
Fleas were present in	2
Bees were present in	2
Sand fleas were present in	1
Snakes were present in	1
Crickets were present in	1
Squirrels were present in	1
Leeches were present in	1

As to *insect bites,* there was no such history in 70 per cent. of 200 cases carefully investigated; a history of mosquito bites in 27 per cent. and in the remainder bites from various insects were recorded.

Any investigation as to the prevalence of an especial insect in a given locality, which is to be of much value, must be made on the spot by a person skilled in entomology, and definite conclusions cannot be based on the above table.

Data as to Illness in Domestic Animals and Birds.

Of 186 families: —

No animal of any kind in	76
Animals present in	110
	186

Thus: —

(*a*) 27 homes had 31 dogs without sickness.
50 homes had 66 cats without sickness.
39 homes had about 745 hens and chickens without sickness.
11 homes had 18 horses without sickness.
3 homes had 3 cows without sickness.
4 homes had 4 canaries without sickness.
10 homes had 2 goldfish without sickness.
1 home had 5 geese without sickness.

1 home had 10 ducks without sickness.
1 home had 10 pigeons without sickness.
(*b*) 6 homes had 14 hens with sickness.
6 homes had 6 hens with sickness.
(*c*) 4 homes had 4 dogs with paralysis.
4 homes had 12 hens with paralysis.
(*d*) 2 homes had deaths in 2 dogs.
6 homes had deaths in 6 cats.
8 homes had deaths in 42 hens.
2 homes had deaths in 2 horses.

Total, 34 homes out of 110 had illness, paralysis or death in 82 animals near the time of the human paralysis.

As to paralysis in domestic animals, a veterinary surgeon, Dr. A. W. May, was employed by the Board in the autumn of 1910 to visit kennels, poultry farms, veterinary hospitals, etc., through as much of the State as possible, in order to see if the distribution of animal paralysis corresponded to that of the human disease. His report will be published separately.

Other data, not given in former reports, follow: —

Nativity.	Cases.
American,	315
French Canadian,	33
French,	51
French American,	5
Irish American,	65
Polish,	19
Portuguese,	13
Hebrew,	15
Italian,	11
English,	9
English American,	2
Irish,	4
French Irish,	2
Colored,	6
Swedish,	15
German American,	6
Greek,	5
Finlander,	2
Nova Scotian,	2
Scotch,	4
Austrian,	1
Bohemian,	1
Not stated,	15
	601

This table is important as showing that the apparent partial immunity of the colored race may be less true than had been supposed, the relative incidence in Massachusetts in 1910 per thousand of population being in the colored race .17, and in the white .20. In order to obtain the incidence among the colored race in a community thoroughly infected, this was calculated for Springfield, where, in a colored population of 1,294, there occurred 4 cases, an incidence of 3.09 per thousand, while in the white population of the same city the incidence was 1.99 per thousand.

Communicability.

The question of communicability is probably obscured by the existence of abortive cases. The data here presented were established with all possible care. In 200 cases there was certain direct contact with an acute case in 32 cases, indirect contact in 10, and direct contact with a possible abortive case in 4. Out of 186 families, 13 had 2 cases and 1 family had 3.

There were no lodgers in 176 families of 186 investigated in this regard, and 18 lodgers in the 10 other families.

Conditions preceding the Attack.

Swimming and *Wading.* — Out of 524 cases when the question was answered, it was stated that 15.8 per cent. of the patients were swimming, wading or paddling in water just before the attack, while the remainder were not.

In 1909, out of 150 cases, 40.8 per cent. gave such a history on account of the different locality investigated last year, which may serve as a reminder that in estimating the possible effect of etiological factors too much importance cannot be attached to investigations confined to one locality.

Exposure to heat, cold or *dampness* just preceding the attack occurred in the history of 27 per cent. of 575 cases and was absent in the rest.

An *accident, fall* or *overexertion* was noted in 23 per cent. of 573 cases just before the attack.

Diet.

The majority of the patients ate of several kinds of food. In the analysis of a series of 200 cases studied in and around Springfield, 4 babies, all under six months, were said to be fed on breast milk alone. The other data as to diet were not apparently of especial interest, many articles of food being given, from a study of which no conclusive data can be drawn.

By Age Periods.

	Cases.	Per Cent. Approximate.
From birth to twelve months, inclusive,	51	8.5
From thirteen months to twenty-three months, inclusive,	65	
Two years old,	61	
Three years old,	98	
Four years old,	69	
Five years old,	51	65.5
	395	
Six to ten years, inclusive,	93	80.8
	488	
Eleven to twenty years, inclusive,	69	92.1
	557	
Twenty-one to thirty years, inclusive,	28	
Thirty-one to eighty years, inclusive,	15	
	600	

It is evident by a comparison with the table for 1909, that in 1910 a larger number of older children were attacked, the percentage of all cases from one to ten inclusive being 87.48 in 1909, while in 1910 only 80.8 per cent. of the same age were attacked.

Mortality by Age.

	Cases.	Deaths.	Mortality (Per Cent.).
Under one year,	38	3	7.89
One to ten years,	451	39	8.64
Over ten years,	112	12	10.71
Total,	601	54	–
Average mortality,	–	–	8.98

The total mortality was 1 per cent. higher than in 1909.

INCIDENCE OF THE DISEASE.

Sex. — There were 331 males and 270 females affected.

The condition of the patient preceding the attack was given in 540 cases; in about 30 per cent. the patient had been perfectly well and in about 60 per cent. some abnormal condition existed either some time before or just previous to the attack. Malaise existed in about 20 per

cent., affection of the throat or respiratory passages in 9 per cent., gastric or intestinal disturbance in 11 per cent. and in the remainder a variety of illnesses, the only group of significance being 10 cases where operation, wounds or sores preceded the attack. A study of the *recent illnesses in members of the same family* showed nothing of apparent importance, a moderate number of ordinary illnesses being recorded, chiefly of the upper respiratory passages and digestive tract.

General Features of Acute Attack for 601 Cases.

540 cases give history of fever.
487 cases give history of pain and tenderness.
284 cases give history of brain symptoms.
209 cases give history of retraction.
92 cases give history of sore throat.
2 cases give history of nystagmus.
1 case gives history of hiccough.

Digestive disturbance during the attack occurred in about 75 per cent. of the cases, this disturbance being nausea and vomiting, constipation or diarrhœa, with colic in a few cases.

Disturbance of the bladder did not occur in 73 per cent. of the cases, but retention was noted in 20 per cent. of the cases and other minor disturbances in the remainder.

Pain and Tenderness.

	Cases.
Pain or tenderness was present in	469
Pain or tenderness was absent in	42
Pain or tenderness was not stated in	90
	601

Pain or Tenderness lasted.

	Cases.
No pain,	42
One day or less,	9
Two days,	14
Three days,	20
Four days,	15
Five days,	10
Six days,	6
One week,	35
One to two weeks,	41
Two to three weeks,	32
Three to four weeks,	18

	Cases.
Four to five weeks,	8
Five to six weeks,	4
Eight to nine weeks,	5
Nine to ten weeks,	1
A few days,	25
Until death,	45
Present when report made,	181
Not stated,	90
	601

Appearance of Paralysis in Days and Weeks after Onset of Fever.

	Cases.
Same day,	20
One day,	31
Two days,	40
Three days,	34
Four days,	15
Five days,	11
Six days,	11
Seven days,	14
Eight days,	4
Nine days,	2
Ten days,	2
Eleven days,	2
Twelve days,	4
Thirteen days,	1
Fourteen days,	1
Two to three weeks,	5
Three to four weeks,	1
Four to five weeks,	1
Eight weeks,	1
	200

Distribution of Early Paralysis.

	Cases.
One leg only,	145
Both legs only,	146
One arm only,	44
Both arms only,	12
One arm and leg, same side,	50
One arm and leg, opposite sides,	18
Both legs and one arm,	32
Both arms and one leg,	8
Both arms and both legs,	51
Ataxia (transitory),	7

	Cases.
Back,	79
Abdomen,	38
Neck,	13
Respiration,	39
Deglutition,	12
Intercostal,	1
Face,	7
Right face,	31
Left face,	24
Strabismus,	2
Not stated,	32

Nine cases out of 200 gave a history of some *skin eruption* appearing on the chest or around the neck, or about the girdle.

In one or two such cases there was noted by the investigator a small maculo-papular rash, — a faint blush underlying the area with no definite redness to base of vesicle.

Prognosis.

Up to the time of assembling the Board's report (a period of six months after the beginning of the epidemic outbreak in western Massachusetts), 27 cases (13.5 per cent.) out of 200 had completely recovered.

A study of the age, distribution of the early paralysis and duration of tenderness and paralysis in these cases leads to the conclusion that the early symptoms offered no means of distinguishing them from cases where the paralysis was to be permanent.

Conclusions.

The disease in Massachusetts was nearly as prevalent in 1910 as in the previous year, and affected 153 cities and towns instead of 136, as in 1909. But one must remember that a much larger proportion of cases is now recognized and reported in Massachusetts than was formerly the case, so that probably the apparently relative prevalence of the disease in Massachusetts when compared to other States, the total number of cases in the State and the apparent spread are in some measure due to the alert attitude of the medical profession of this State in recognizing and reporting cases.

A large epidemic center existed in Springfield, with 148 cases in the city and a large number in the surrounding towns, the distribution appearing to be radial from Springfield. Another epidemic center existed in Fall River, with 89 cases in the city and more or less radial distribution to contiguous towns.

It is evident that the disease has existed in all classes in the community,

as is evident from the report on the sanitary conditions under which the patient lived, and also that it exists under all conditions of sewage disposal and with all kinds of water supply. Our researches in the last two years have failed to show an excessive amount of dust in affected localities.

The proportion of affected houses in which contemporaneous sickness, paralysis or death existed in domestic animals or birds still seems larger than one would naturally expect.

The occurrence of 6 cases in the colored race is of importance in connection with the data of other investigators, who have found the race rarely affected.

The facts given with regard to communicability are to be regarded as important, 42 such histories having been obtained in 200 cases.

No definite information as to any one factor is to be found in the antecedents of the attack, since bathing, falls, exposure to heat, overexertion, etc., are common occurrences in children of the affected age in the summer season; nor in the study of diet does there seem much of importance except for the existence of the disease in 4 nursing babies.

The fact has been commented on that the disease was a little more prevalent this year in older children than in 1910.

The study of the immediate antecedents of the attack and the early symptoms show nothing of especial present interest except the very common occurrence of pain and tenderness, and the information as to its duration is of value.

The distribution of paralysis was made by a skilled investigator in a group of cases carefully studied, and it is important to note under those conditions the comparatively frequent involvement of the back, abdomen, neck and face, — a matter often overlooked.

The per cent. of total recoveries from the paralysis within a period of six months and less after the attack was 13.5 in 200 cases, as against a per cent. of 16.7 in a similar class of cases in 1909.

For the year 1911 the State Board of Health intends to pursue the same investigation as in the four previous years, on a more extended scale, hoping by a study of the disease in one locality over a term of years to reach some conclusions as to its characteristics.

As in former years the Board is under great obligations to the medical profession for their ready co-operation at all points, and asks for the coming year the same ready assistance.

ANTERIOR POLIOMYELITIS.[1] — ATTEMPTS TO TRANSMIT THE DISEASE TO MONKEYS BY INOCULATION WITH THE NASAL, PHARYNGEAL AND BUCCAL SECRETIONS OF EIGHTEEN HUMAN CASES.

By M. J. Rosenau, M.D., Professor of Preventive Medicine and Hygiene, Harvard Medical School, Boston; Philip A. E. Sheppard, M.D., Special Investigator for the Massachusetts State Board of Health, Boston; and Harold L. Amoss, M.D., Assistant in Preventive Medicine and Hygiene, Harvard Medical School, Boston.

We lack definite information concerning the mode of transmission of epidemic poliomyelitis. Such information is of fundamental importance from the practical standpoint of preventing the spread of the disease. Flexner and Lewis have shown that the virus may be obtained in the nasal and pharyngeal mucosa of monkeys, and it has been assumed that the virus leaves the body in the secretions of the mouth and the nose; the mode of entrance is perhaps through the nasal and pharyngeal mucosa. This view is strengthened by the known facts in a somewhat analogous infection, — epidemic cerebro-spinal meningitis.

We have undertaken this work to determine whether the virus of epidemic poliomyelitis may be demonstrated in the nasal, pharyngeal and buccal secretions of patients in various stages of the disease, in convalescents, or in suspected carriers.

The methods employed were as follows: —

Collection of Material.

The specimens were all personally obtained by one of us (P. A. E. S.). The patient was taken just as he was found, without being cleaned up at all, but the usual antiseptic precautions were employed by the operator, and all instruments and appliances were sterilized.

Each specimen was collected in a sterile vial. The salt solution used to wash the mucous membranes of the nose and throat was practically normal and was freshly made for each case. The instruments used were a Goodale syringe, a nasal douche, nasal forceps and applicators.

To secure the specimen the patient was seated directly before the operator and first instructed to gargle with a mouthful of salt solution, which was then secured in a sterile bottle; next the nasal douche was filled with salt solution and used alternately in each nares as follows: —

With the patient's head slightly tipped back, until the floor of the nares

[1] Report to the State Board of Health of Massachusetts, February, 1911. Reprinted from "Boston Medical and Surgical Journal," May 25, 1911.

is approximately horizontal, the salt solution is allowed to flow in gently. The douche is then removed, the nares closed with the thumb and finger of the operator. The head of the patient is next brought forward as far as necessary to allow the salt solution to run out through the opposite nares (this is brought about by releasing pressure on the appropriate side), and the fluid is caught in the sterile bottle. In order to expel the last portions of salt solution, and at the same time to remove the residual mucus and nasal secretion, the patient is instructed to blow gently through the open nostril. The procedure is then repeated on the other side. The applicator, tipped with sterile absorbent cotton, is used repeatedly along the floor of the nasal cavities and gently passed back into the pharynx in order to clean up and remove those portions of mucous and nasal secretions which cannot be obtained by simple irrigation. The mouth is now swabbed with sterile absorbent cotton; or, if the patient is old enough to understand, he is instructed to chew the pledget of absorbent cotton, this being done in order to secure the saliva. If the tongue is at all coated, some of this material is wiped off as well.

The Goodale syringe, filled with salt solution, is now introduced into the mouth and passed back gently until the tip is inserted under the uvula, and directed upward and forward into the pharyngeal vault, when the contents of cylinder (containing about 3 cubic centimeters salt solution) is expressed quickly and the syringe withdrawn. The vestibules of the nasal cavities having previously been plugged with sterile absorbent cotton, the head of the patient is lowered immediately, to allow the salt solution to drain into the absorbent cotton. These pledgets of cotton are added to the other secretions in the sterile bottle, and the patient is instructed to blow gently, first through one, then through the other nostril, into the collecting bottle. The applicator, tipped with cotton, is again brought into play in order to clean up the cavities still further. This process is repeated two or three times in each case, until the cavities are fairly well cleared out, the end in view in every case being to secure a large amount of material with as little discomfort as possible to the patient.

Experimental Technic.

The material thus collected was cooled and brought without delay to the laboratory, packed in ice. It was then passed through a Berkfeld filter, warmed to about 30° C., and injected into the brain and peritoneal cavity of monkeys (Macacus Rhesus).

A small gimlet hole was bored in the skull on each side over the frontal lobes (under ether anesthesia). About 4 cubic centimeters of the filtrate was then introduced into each cerebral hemisphere and the remainder was injected into the peritoneal cavity.

A brief account of the 18 cases follows: —

Case No. 1.

F. F., age five, male, American, living in Springfield, Mass. Sanitary condition of dwelling good. No other case in the family. The boy appeared to be well developed and nourished, and, the mother says, was always healthy.

Acute febrile attack Sept. 25, 1910, followed by paralysis of the muscles of deglutition and vocal cords, with aphonia on the sixth day.

The general features of the acute attack were nausea and vomiting, starting on the morning of September 25, and recurring at intervals during the next twenty-four hours, temperature of 103° F., restlessness and wandering of mind, pain and tenderness lasting a few days in the neck muscles and right leg, sore throat and aphonia, persistent constipation.

On September 13 (twelve days before the acute febrile attack), tonsils and adenoids were removed at a Springfield hospital.

The boy was first seen by one of us early in October, 1910. He was in a very nervous condition and all reflexes were exaggerated. He could swallow with difficulty, though his voice was returning. The throat was slightly injected and there was some bronchial cough.

On November 5, when the specimen was secured for inoculation, he was still very nervous, and the throat symptoms persisted, but the paralysis had practically disappeared.

Monkey (1). — On Nov. 5, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 50 cubic centimeters into peritoneal cavity. Normal recovery from the anesthesia. Never showed any suspicious symptoms, though on November 11 temperature was 103.3° F. Dismissed from observation Feb. 2, 1911.

Case No. 2.

D. Y. (American born of Scotch parents), age five, living in Ludlow, Mass. Sanitary condition of dwelling good. No other case in family. Had scarlet fever and was in Springfield Isolation Hospital from Aug. 6 to Sept. 24, 1910, a period of seven weeks, during which time there were cases of anterior poliomyelitis in the same isolation hospital. During his stay there the child had a bad sore throat and several cultures were taken, all of which proved to be negative for the diphtheria bacillus.

Patient returned to Ludlow September 24, and two weeks later (October 7) acute febrile attack developed, followed two days later by paralysis of right arm and both legs.

The general features of acute attack were: nausea and vomiting, fever, 102° F., retraction of head, brain symptoms (listless and stupid), pain in head, which had lasted two days when paralysis of right arm and both legs appeared. Urine was negative. Vomiting and diarrhœa accompanied the febrile attack, and the diarrhœa continued for several weeks, being followed finally by constipation.

On November 5 (four weeks after his febrile attack) specimen was secured for inoculation. The child was still in bed, with a temperature of

101° F. There was bronchial cough, the throat was slightly injected and paralysis of both legs and right arm persisted.

Monkey (*2*). — On Nov. 5, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 85 cubic centimeters into peritoneal cavity. The animal recovered promptly from the operation and never showed suspicious symptoms. November 10, temperature 104.8° F. Dismissed from observation Feb. 1, 1911.

Case No. 3.

V. P., age four, female, born of American parents, living in Springfield, Mass. Well developed and nourished. Sanitary condition of dwelling good. No other case in the family.

Acute attack Oct. 11, 1910, with fever, pain and tenderness along spine. Paralysis developed seventy-two hours later, involving both legs.

On November 5, when specimen was obtained for inoculation, the child was in good health; the walk was ataxic.

Monkey (*3*). — On Nov. 5, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 50 cubic centimeters into the peritoneal cavity. Monkey recovered well from the anesthesia and remained lively and apparently well. Dismissed from observation Feb. 1, 1911.

Case No. 4.

T. C. (American born of Irish parents), age eighteen, female, living in Springfield, Mass. Sanitary condition of dwelling good. No other case in the family. T. C. well developed and before onset was well nourished.

Acute febrile attack occurred Aug. 18, 1910, and the next day paralysis of both legs, both arms, back and abdomen developed.

The general features of the acute attack were sudden fever, 104° F., intense pain and tenderness in the back and lower extremities, lasting six weeks, together with marked change of temperament, — irritability. On the second day of attack patient voided a clear green urine with a trace of albumen.

On November 5, when the specimen was taken, the paralysis of both legs and both arms still persisted, and there was slight involvement of intercostals, so that she could not cough properly. She was apathetic and weak, but had recovered in large measure her sunny disposition.

Monkey (*4*). — On Nov. 5, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 75 cubic centimeters into peritoneal cavity. Recovered promptly from the operation, but on November 7 (two days later) was unusually quiet, did not climb and hid his face. The next day (November 8) the temperature was 102.5° F., and he was placed in a separate cage. On November 9 his disposition seemed changed, he did not express fear and could be approached without being excited. No paralysis; walked on four legs. On November 10 he seemed weak and dazed, and temperature was 101.4° F. On November 11 would not eat except when fed by hand; pupils equal, reacted to light; temperature, 100.4° F. Hand

and wrist on right side weaker. November 12, general weakness, moved in circles to the left, head and eyes turned to the left; temperature, 98° F. Died November 13, 3 P.M. Autopsy showed a hemorrhage in the right anterior lobe in the white matter, with pus in the left brain near the motor area for the arm. Cortex was markedly congested, the dura apparently normal.

Case No. 5.

R. T., age five, male (American born of Irish parents), living in Springfield, Mass. Sanitary condition of dwelling bad. Patient well developed and nourished. No other case in family.

Acute attack Oct. 6, 1910, with high fever and sore throat. Paralysis of both legs developed a week later.

On November 5, when specimen was obtained for inoculation, paralysis was disappearing. Tonsils were still swollen, however, and there was some bronchial cough.

Monkey (*5*). — On Nov. 5, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 65 cubic centimeters into peritoneal cavity. The animal recovered promptly from the anesthetic, but the next morning did not use right arm, which hung limp; tendency to lean towards right. November 7, both the right arm and the right leg were weak; would not eat. On November 9 he ate better and used right leg somewhat, but right arm was still limp; temperature, 102° F. The condition improved somewhat, but the lameness of right arm and right leg persisted. It is most probable that the paralysis was the result of mechanical injury at the time of the operation. Dismissed from observation Feb. 2, 1911.

Case No. 6.

A. L. T., age five, male, American, living in Middleborough, Mass. Sanitary condition of dwelling good. No other case in the same family, but younger brother was sick with dysentery about the same time. Lived an out-of-door life. For a couple of weeks before onset was fussy and irritable.

Acute attack Oct. 25, 1910, with the following general features: fever ranging from 100° to 104° F., delirium, pain and tenderness general, but more pronounced in legs; disturbance of vision and speech lasting two days; inclined to jump and twitch; reflexes absent. Paralysis appeared two days after onset, involving the left leg.

On November 13, when specimen for inoculation was obtained, patellar reflex was absent. Paralysis of left leg persisted. General condition improved.

Monkey (*6*). — On Nov. 13, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 50 cubic centimeters into peritoneal cavity. Recovered promptly from the operation. On November 15 developed nervous symptoms, with irritability, and nodding of head when disturbed. Was held by the other monkeys as though ill. The nervous symptoms gradually disappeared, but the monkey became weaker, — refused

to eat. Chloroformed November 9. Autopsy showed an old tuberculous lesion of the lung, with a generalized tuberculosis throughout the abdominal organs.

Case No. 7.

E. H. W., age eight, male (American born of Scotch parents), living in Middleborough, Mass. Sanitary condition of dwelling good. No other case in the family at this time. Patient well developed and nourished.

Acute attack Sept. 17, 1910, preceded by vomiting, accompanied by fever (102° F.), frontal headache, pain and tenderness on left side, also right shoulder and arm, coryza, cough, constipation. Paralysis appeared five days later, involving the left leg. Slight difficulty in voiding urine.

On November 13, when the specimen was obtained for inoculation, patient was but slightly ataxic in his gait, paralysis was disappearing and general condition was distinctly on the mend.

Monkey (7). — On Nov. 13, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 75 cubic centimeters into peritoneal cavity. Recovered well from the operation and soon began to climb and jump. November 15 exhibited trembling or shivering motions. Otherwise negative. Dismissed from observation Feb. 2, 1911.

Case No. 8.

K. J. W., age ten years, male (American born of Scotch parents), living in Middleborough, Mass. Older brother to the preceding case. Sanitary condition of dwelling good. In intimate contact with his brother during the course of his acute illness. Felt well, however, up to the day of acute attack, which occurred Nov. 3, 1910, with high fever, accompanied by pain and tenderness in back of neck, lasting forty-eight hours. There were also gastro-enteritis and vomiting. Paralysis of right leg was noticed on fourth day.

On November 13, when specimen was obtained for inoculation, leg was still paralyzed, the tonsils were injected and there were coryza and cough.

Monkey (8). — On Nov. 13, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 50 cubic centimeters into peritoneal cavity. Recovered promptly from the operation, and nothing abnormal was noted subsequently. Dismissed from observation Feb. 2, 1911.

Case No. 9.

A. T. McE., age six years, female (American born of Irish parents), living in North Easton, Mass. Sanitary condition of dwelling fair. No other case in family. Onset Nov. 21, 1910, with coryza, fever, pain and tenderness in abdominal region. Paralysis of both legs appeared three days later.

On November 25, when specimen was obtained for inoculation, patellar reflexes were absent. Patient was unable to walk. Hyperesthesia and pain along spine were decreasing. Throat injected, tonsils swollen.

Gave history of having passed two round worms a few weeks before the acute onset.

Monkey (9). — On Nov. 25, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 70 cubic centimeters into peritoneal cavity. Recovered promptly from the operation. On December 1 had a suspicious way of lifting the left forearm and wrist. This, however, soon passed away. Dismissed from observation Feb. 2, 1911.

Case No. 10.

Dr. R. (age about thirty-five), American, living and practicing in Springfield, Mass. Sanitary condition of dwelling excellent.

Specimen was taken from his nose and throat on Dec. 5, 1910, for the reason that he had seen and been associated with several cases of infantile paralysis. The object was to determine whether or not such persons could possibly act as carriers.

At the time of taking this specimen the doctor was in good health, but had a slight coryza.

Monkey (10). — On Dec. 5, 1910, 2 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 65 cubic centimeters into peritoneal cavity. Recovered well from the operation, but on second day trembled as though shivering from the cold. This symptom soon passed away, however. In a few days temperament seemed somewhat altered, was less wild, but no other suspicious symptoms developed. Dismissed from observation Feb. 1, 1911.

Case No. 11.

H. R., age three years, female, well developed and nourished, living in Springfield, Mass. Sanitary condition of dwelling excellent. Living at home with her father, a practicing and school physician.

A younger sister, age nine months, had an attack of poliomyelitis on July 15, 1910, resulting in a complete right hemiplegia.

Acute attack Nov. 23, 1910, with gastro-enteritis, vomiting, fever and pain in her left face. The next day paralysis of the left face was noticed.

On December 5, when the specimen for inoculation was secured, paralysis of the left face persisted, throat was injected and tonsils were enlarged. Otherwise, general condition of health good.

Monkey (11). — On Dec. 5, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 60 cubic centimeters into peritoneal cavity. On December 6 seemed to be lame in right leg and had a tendency to fall to the right side. Pupils equal and reacted to light. Did not exhibit usual fear and refused to eat. On December 7 seemed delirious and moved continually in circles to the right. The condition gradually improved so that by December 14 the monkey climbed and jumped much as usual, but still exhibited lameness of right arm. Chloroformed December 21, and near the right motor area was found a blood clot, which accounted for the paralysis.

Case No. 12.

M. B., age twenty-one, female, American, living in Springfield, Mass. Sanitary condition of dwelling good. No other case in family. Library assistant, well developed and nourished.

Acute attack Dec. 4, 1910. Slight rise of temperature, marked change of temperament, pain and tenderness in the neck, paralysis of right face and neck. For three or four weeks before onset, constipation, which still persists.

On December 8, when specimen was obtained for inoculation, paralysis of right face was still present. Throat injected, tonsils hypertrophied, adenoids present, lower turbinates swollen, injected and bleed easily.

Monkey (*12*). — On Dec. 8, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 60 cubic centimeters into peritoneal cavity. Recovered promptly from the anesthetic and never showed suspicious symptoms. Dismissed from observation Feb. 2, 1911.

Case No. 13.

S. S., age two years three months, male, Hebrew, living in Chelsea, Mass. Sanitary condition of dwelling fair.

Acute attack Dec. 10, 1910, with fever, coryza, bronchial cough, pain and tenderness in right knee. Paralysis appeared on the same day, involving the right leg. Child well developed and nourished.

On December 15, when specimen was obtained for inoculation, paralysis had disappeared.

Monkey (*13*). — On Dec. 15, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 20 cubic centimeters into peritoneal cavity. (In disconnecting the filter, the cotton plug was sucked in, and for fear of contamination this material was again passed through a Berkfeld filter.) Recovered promptly from the anesthesia and never showed suspicious symptoms. Dismissed from observation Feb. 2, 1911.

Case No. 14.

A. B., age twelve months, female, French-Canadian, living in Lawrence, Mass. Sanitary condition of dwelling poor.

Acute attack Dec. 8, 1910, with fever and sore throat. Three weeks previously, the child had spells of vomiting lasting for two days, and was generally peevish, then seemed to be in good health up to date of onset. Paralysis involving the left arm appeared on December 9.

On December 15, when specimen was secured, child was in fair health, except for the paralysis. She was constipated, however, and throat was sore and injected, tonsils were enlarged and reddened, and there was some cough.

Monkey (*14*). — On Dec. 16, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 20 cubic centimeters into peritoneal cavity. Recovered slowly but completely from the operation, and never showed suspicious symptoms. Dismissed from observation Feb. 2, 1911.

Case No. 15.

M. F. B., age nine months, female (American born, French-Canadian parents), living in Wakefield, Mass. Sanitary condition of dwelling fair. No other case in family. A well developed and nourished child. Breast fed until one month before onset, when she was partially weaned and given cow's milk and baked potatoes.

Acute attack, Dec. 15, 1910, with fever, brain symptoms, vomiting, convulsions, pain and tenderness. Paralysis of right leg, right arm and right face appeared two days later.

On December 18, when specimen was secured, the child was unconscious. She died on December 20. Autopsy refused.

Monkey (*15*). — On Dec. 19, 1910, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 50 cubic centimeters into peritoneal cavity. Recovered promptly from the operation and never showed suspicious symptoms. Dismissed from observation Feb. 2, 1911.

Case No. 16.

T. G., age twenty-three years, male, Italian, living in Southbridge, Mass. Sanitary condition of dwelling fair. Patient well developed but poorly nourished.

Acute attack Dec. 27, 1910, with fever, pain and tenderness on the right, a week later extending to the left side, along the spine and the extremities, profuse sweating and evidences of vasomotor change of lower extremities.

Paralysis appeared at time of onset, and involved at first only the thumb and index finger of the right hand. The next day paralysis invaded the right forearm and arm, thence extending to the right leg, where it was complete. It lasted only two days, however, during which period no reflexes could be obtained.

January 10, when the specimen was secured, all the reflexes were exaggerated on the right, and diminished on the left side. There was no rise of temperature. Tongue clean, tonsils enlarged, soft palate and uvula congested and slightly edematous, noticeable change of temperament.

Monkey (*16*). — On Jan. 10, 1911, 3 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 30 cubic centimeters into peritoneal cavity. Recovered slowly from the anesthesia and never showed suspicious symptoms. Dismissed from observation February, 1911.

Case No. 17.

G. S., age seven years, female, American, living in Ware, Mass. Sanitary condition of dwelling excellent. No other case in the family. Attended public school and there occupied a seat immediately behind a boy who, just previous to her attack, was out of school with poliomyelitis.

Onset Dec. 11, 1910. No fever. Paralysis appeared the same day, involving both legs. Pain and tenderness, more pronounced in the thighs, were

a marked feature. Hiccough frequent and persistent since attack. For a week or ten days before her attack the mother remarked a peculiar change of temperament.

On Jan. 12, 1911, when specimen was secured, she was completely recovered, throat normal, tonsils slightly hypertrophied, otherwise a normal child.

Monkey (*17*). — On Jan. 13, 1911, 4 cubic centimeters were injected into right brain, 4 cubic centimeters into left brain, and 30 cubic centimeters into peritoneal cavity. Recovered promptly from the operation and never showed suspicious symptoms. Dismissed from observation February, 1911.

Case No. 18.

W. C., age nineteen months, male, French-American, living in Ware, Mass. Sanitary condition of dwelling fair. No known history of contact with any other case.

Acute attack on Jan. 1, 1911, with vomiting, accompanied with convulsions and diarrhœa, fever, stupor, retraction of head, rigidity of right arm and right leg. Paralysis followed eight days later, involving right arm and right leg.

On Jan. 12, when specimen was secured, the baby was moribund, reflexes were absent on both sides, though the child could move its legs and arm. Nystagmus, tongue heavily coated, throat injected, tonsils hypertrophied. Died on Jan. 18, 1911. Autopsy refused.

Monkey (*18*). — On Jan. 13, 1911, injected 4 cubic centimeters into right brain, 4 cubic centimeters into left brain, and 10 cubic centimeters into peritoneal cavity. Recovered promptly from the anesthesia and never showed suspicious symptoms. Dismissed from observation February, 1911.

Summary and Conclusions.

The fact that the virus of epidemic poliomyelitis was not demonstrated in the buccal and nasal and pharyngeal secretions of these 18 cases is not proof that it may not have been there. In any event, the virus would have been greatly diluted, owing to the relatively large amount of salt solution used to wash the mucous membrane. Furthermore, under the conditions of the experiment the virus may have been so attenuated as to be incapable of infecting monkeys.

It is well known that a considerable quantity of the virus from the central nervous system of man is sometimes required to initiate the infection in monkeys.

Our results must not be construed as in any sense disproving the assumption that the infection in this disease may be discharged from the mucosa of the upper respiratory passage and enter through the same channel.

	Epidemiological and Clinical Data.						*Experimental Data.*			
Case.	Condition before Onset.	Date of Onset.	Character of Onset.	Date of Paralysis.	Extent of Paralysis.	Condition of Patient when Specimen taken.	Time after Onset when Specimen taken.	Monkey.	Amount of Filtrate injected, and Sites.	Results.
F. F. Male. Age, five years.	Sore throat.	Sept. 25, 1910.	Gastro-intestinal upset, with fever.	Oct. 1, 1910.	Muscles of deglutition and vocal cord. Complete aphonia.	Nervous, sore throat, paralysis disappeared.	Six weeks (*i.e.*, on Nov. 5, 1910).	No. 1.	4 ccm. Right brain. 4 ccm. Left brain. 50 ccm. Peritoneum.	Negative.
D. Y. Male. Age, five years.	Sore throat. Convalescing from scarlet fever.	Oct. 7, 1910.	Gastro-intestinal upset, with fever.	Oct. 9, 1910.	Complete. Right arm and both legs.	Complete paralysis right arm and both legs, fever, cough and weak.	Four weeks (*i.e.*, on Nov. 5, 1910).	No. 2.	4 ccm. Right brain. 4 ccm. Left brain. 85 ccm. Peritoneum.	Negative.
V. P. Female. Age, four years.	In good health.	Oct. 11, 1910.	Febrile.	Oct. 13, 1910.	Complete of both legs.	Recovering from paralysis, walks, slightly ataxic gait.	Three weeks (*i.e.*, on Nov. 5, 1910).	No. 3.	4 ccm. Right brain. 4 ccm. Left brain. 50 ccm. Peritoneum.	Negative.
T. C. Female. Age, eighteen years.	In good health.	Aug. 18, 1910.	Sudden chill. Fever. No prodromal symptoms.	Aug. 19, 1910.	Both legs and both arms, back, abdomen, intercostals.	Paralysis persists, atrophy.	Six weeks (*i.e.*, on Nov. 5, 1910).	No. 4.	4 ccm. Right brain. 4 ccm. Left brain. 75 ccm. Peritoneum.	Paralysis right arm. Died seventh day. Abscess in left motor area.
R. T. Male. Age, five years.	In good health.	Oct. 6, 1910.	Febrile.	Oct. 20, 1910.	Both legs.	Coryza.	Four weeks (*i.e.*, on Nov. 5, 1910).	No. 5.	4 ccm. Right brain. 4 ccm. Left brain. 65 ccm. Peritoneum.	Paralysis right arm (mechanical).

A. L. T. Male. Age, five years.	Irritable for two weeks.	Oct. 25, 1910.	Febrile. Delirium and pain.	Oct. 27, 1910.	Left leg.	Paralysis persists. Reflex absent.	Two weeks (*i.e.*, on Nov. 13, 1910).	No. 6.	4 ccm. Right brain. 4 ccm. Left brain. 50 ccm. Peritoneum.	Nervous symptoms on third day, became very weak and died on twenty-fifth day. Generalized tuberculosis.
E. H. W. Male. Age, eight years.	In good health.	Sept. 17, 1910.	Febrile. With pain and vomiting; coryza.	Sept. 22, 1910.	Left leg.	Paralysis disappearing, ataxic gait improving.	Seven weeks (*i.e.*, on Nov. 13, 1910).	No. 7.	4 ccm. Right brain. 4 ccm. Left brain. 75 ccm. Peritoneum.	Negative.
K. T. W. Male. Age, ten years.	In good health.	Nov. 3, 1910.	Febrile. With gastro-enteritis; pain.	Nov. 7, 1910.	Right leg.	Paralysis persists. Coryza and cough.	Ten days (*i.e.*, on Nov. 13, 1910).	No. 8.	4 ccm. Right brain. 4 ccm. Left brain. 50 ccm. Peritoneum.	Negative.
A. T. McE. Female. Age, six years.	Passed two round worms two weeks before attack.	Nov. 21, 1910.	Febrile. With coryza and pain.	Nov. 23, 1910.	Both legs.	Paralysis persists. Reflex absent, pain diminished, throat injected, tonsils swollen.	Five days (*i.e.*, on Nov. 25, 1910).	No. 9.	4 ccm. Right brain. 4 ccm. Left brain. 70 ccm. Peritoneum.	Negative.
Dr. R.[1] Male. Age, thirty-five years.	In good health.						Dec. 5, 1910.	No. 10.	2 ccm. Right brain. 4 ccm. Left brain. 65 ccm. Peritoneum.	Negative.
H. R. Female. Age, three years.	In good health.	Nov. 23, 1910.	Febrile. With pain.	Nov. 24, 1910.	Left face.	Paralysis persists, throat injected, tonsils enlarged.	Twelve days (*i.e.*, on Dec. 5, 1910).	No. 11.	4 ccm. Right brain. 4 ccm. Left brain. 60 ccm. Peritoneum.	Paralysis right leg and arm, brain symptoms. Chloroformed sixteenth day. Blood clot in left motor area.

[1] Suspected carrier.

Epidemiological and Clinical Data — Concluded. *Experimental Data* — Concluded.

Case.	Condition before Onset.	Date of Onset.	Character of Onset.	Date of Paralysis.	Extent of Paralysis.	Condition of Patient when Specimen taken.	Time after Onset when Specimen taken.	Monkey.	Amount of Filtrate injected, and Sites.	Results.
M. B. Female. Age twenty-one years.	In good health.	Dec. 4, 1910.	Febrile. With marked change of temperament.	Dec. 4, 1910.	Right face.	Paralysis persists, pain.	Fourth day (*i.e.*, on Dec. 8, 1910).	No. 12.	4 ccm. Right brain. 4 ccm. Left brain. 60 ccm. Peritoneum.	Negative.
S. S. Male. Age, two years, three months.	In good health.	Dec. 10, 1910.	Febrile. With coryza.	Dec. 10, 1910.	Right leg.	Cough, coryza. Paralysis disappeared.	Fifth day (*i.e.*, on Dec. 15, 1910).	No. 13.	4 ccm. Right brain. 4 ccm. Left brain. 20 ccm. Peritoneum.	Negative.
A. B. Female. Age, twelve months.	Vomiting and peevish.	Dec. 8, 1910.	Febrile.	Dec. 9, 1910.	Left arm.	Paralysis persists.	Seventh day (*i.e.*, on Dec. 15, 1910).	No. 14.	4 ccm. Right brain. 4 ccm. Left brain. 20 ccm. Peritoneum.	Negative.
M. F. B. Female. Age, nine months.	Fretful, otherwise well.	Dec. 15, 1910.	Febrile. Vomiting and convulsions.	Dec. 17, 1910.	Right leg, right arm, right face.	Fever, convulsions. Paralysis persists.	Fifth day (*i.e.*, on Dec. 20, 1910).	No. 15.	4 ccm. Right brain. 4 ccm. Left brain. 50 ccm. Peritoneum.	Negative.
T. G. Male. Age, twenty-three years.	In good health.	Dec. 27, 1910.	Febrile.	Dec. 27, 1910.	Right arm; later, right leg.	Vasomotor disturbance, throat injected.	Two weeks (*i.e.*, on Jan. 10, 1911).	No. 16.	3 ccm. Right brain. 4 ccm. Left brain. 30 ccm. Peritoneum.	Negative.
G. S. Female. Age, seven years.	In good health.	Dec. 11, 1910.	Sudden. No fever.	Dec. 11, 1910.	Both legs.	In good health. Tonsils hypertrophied.	Four weeks (*i.e.*, on Jan. 12, 1911).	No. 17.	4 ccm. Right brain. 4 ccm. Left brain. 30 ccm. Peritoneum.	Negative.
W. C. Male. Age, nineteen months.	In good health.	Jan. 1, 1911.	Fever, stupor, retraction of head.	Jan. 8, 1911.	Right arm and right leg.	Moribund.	Eleven days (*i.e.*, on Jan. 12, 1911).	No. 18.	4 ccm. Right brain. 4 ccm. Left brain. 10 ccm. Peritoneum.	Negative.

AN INVESTIGATION AS TO THE OCCURRENCE IN MASSACHUSETTS OF PARALYSIS IN THE LOWER ANIMALS AND FOWLS.[1]

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The investigation of animal paralysis was made from Oct. 15 to Dec. 31, 1910, and consisted mainly of interviews with veterinarians, poultry raisers and dog breeders. The area covered during the investigation included the whole of Berkshire, Middlesex and Essex counties and parts of Franklin, Worcester, Norfolk, Suffolk and Plymouth counties.

The cases detailed in this report are all of the cases of paralysis reported except those associated with azoturia, parturient paresis and the paralysis of dogs subsequent to distemper.

No. 1.

Needham. — Dr. S. O. Fowle. Horse, thirty-two years old. Paraplegia developed in the stable. Destroyed within forty-eight hours.

No. 2.

Winchester. — Dr. Wm. Buckley. Several cases of paralysis in old livery horses. Paraplegia developed while the horses were at work and also in the stable. Destroyed.

No. 3.

North Adams. — Dr. A. A. McDonell. Numerous cases of paralysis (paraplegia) in horses from 1892–1902. Prevalent in winter. No cases in the last few years. Four cases seen, several years ago, at a farm on Florida Mountain. These cases all occurred in colts fed on wild grasses, and no cases have developed since the farm was ploughed and seeded down. Symptoms, as remembered, were swaying of hind quarters, staggering gait, capricious appetite. Bowels normal. No atrophy noticed after recovery; 70 per cent. recovered. "Has been prevalent in Vermont and has been investigated." The investigation in Vermont resulted in the opinion that the paralysis was caused by feeding foxtail, one of the wild grasses.

No. 4.

North Adams. — Dr. R. T. Williams. In August, 1910, 3 pigs four to five months old were seen at a farm on the road to Savoy, over Burlingame Hill. Paraplegia; sudden onset. Recovered.

[1] An investigation made under the direction of the Massachusetts State Board of Health to determine, in a general way, the frequency of paralysis in the lower animals. Whether or not such animal paralyses have any relation to infantile paralysis as seen in human beings has not yet been determined, but will be the subject of extended investigation during 1911 and 1912.

No. 5.

Gardner. — Dr. A. S. Cleaves. Red roan gelding, eleven years old. The case first visited by Dr. Cleaves the last of April or first of May, 1909. Horse had been working on farm for four or five days hauling manure from barn to field. Work was not excessive. Horse became suddenly paralyzed, fell on the road, and was removed to stable on a stone boat. The symptoms were characteristic of azoturia, but the urine was normal in color and consistency. The paralysis was not complete as the horse was able to get up, with some difficulty, during the following few days. The case was not seen by veterinarian until three weeks later. The paralysis and loss of co-ordination were very pronounced. The patient was in the field, and when forced to trot swayed and fell down, finally getting up on fore feet and dragging hind legs. The case was visited with Dr. Cleaves Oct. 27, 1910. The horse was trotted, and showed a peculiar rolling motion in the hind quarters, inability to take the normal length of stride, and loss of co-ordination. The horse was in poor condition, but showed no distinct atrophy of any one set of muscles.

No. 6.

Westminster. — Poultry Farm A. Thirty or more cases of chicken paralysis have occurred on this farm during the past season. The chickens were turned into the orchard in June, a week after the spraying with arsenate of lead had been finished. The orchard contained plenty of grass and fallen apples, on which the chickens fed. The disease appeared in September and still existed in October, although the fowls had been removed to their winter houses. The fowls affected were all five months old, no cases occurring among the older stock. The first symptom was slight drooping of one wing, and then in forty-eight hours the wing and leg on the same side would become completely paralyzed. The appetite remained good during the disease. The fowls were destroyed as soon as the paralysis became complete. No cases in 1909.

No. 7.

Westminster. — Poultry Farm B. This farm adjoins the farm noted in the previous report (No. 6). During the past season there have been 10 cases of chicken paralysis among the five-months-old fowls, and at the present time (Oct. 27, 1910) there are 4 new cases. The chickens presented the same symptoms, and were kept in the orchard, as in the previous case. The only difference in the conditions of the two farms was that in the latter the orchard was not sprayed. The drainage from the orchard on farm A runs directly away from premises B.

No. 8.

Westborough. — Dr. Chas. H. Reed. A cow, owned by W. E. G., became suddenly paralyzed (paraplegia) in pasture Aug. 21, 1910. Driven with difficulty to barnyard. Due to calve Jan. 13, 1911. Symptoms August 21:

paraplegia, volar flexion, head extended and turned slightly on its axis. Appetite fair; bowels normal. October 31, cow down; motor and sensory paralysis complete in hind quarters; head extended and turned, right horn being lower than the left.

No. 9.

Westwood. — Dr. J. J. Mulvehill. Paraplegia in a cat. Case was visited but once and final result not known.

No. 10.

Dedham. — Dr. Edward Knobel. July, 1910. A French bulldog; sudden attack paraplegia, gradually working forward, followed by death.

No. 11.

Pittsfield. — Dr. E. L. Hannon. *A.* Three mongrel puppies, three months old, affected with paraplegia Aug. 29, 1910. Paralysis increased; dogs died in a few days. Owned in Lebanon, N. Y.

B. January, 1910. Gelding, twelve years old. Patient was found down in stall in the morning, unable to rise; paraplegia. Temperature 103.2°; urine high colored. Horse destroyed.

No. 12.

Pittsfield. — Dr. J. A. Brackin. *A.* Eight or ten years ago numerous cases of paralysis (principally paraplegia) in colts. Five or six colts have been seen affected on one farm at one time. "Mr. C. A. of Richmond had a colt paralyzed in July, but it is now better and being driven, although stiff gaited. Mr. A. has had 2 cases of anterior poliomyelitis in his children in the last three years."

B. A large number of cases of paraplegia in colts seen in New York State a number of years ago.

No. 13.

Cohasset. — Dr. D. W. Gilbert. Cat owned by W. J. C., North Cohasset. Paralyzed (paraplegia) November 7. Recovering slowly.

No. 14.

Newtonville. — Dr. W. T. White. French bull dog; paraplegia. Recovered in three days.

No. 15.

Brockton. — Dr. W. H. Brownell. Gray mare; became paralyzed in hind quarters on the road, August 16. Case was typical of azoturia, but there was no apparent change in the urine. The horse improved gradually, but showed considerable paralysis November 30.

No. 16.

Brockton. — Dr. B. D. Curtis. Severe outbreaks of pig paralysis during the past few years.

No. 17.

Brockton. — Dr. J. K. Mason, Campello. *A.* March, 1910. Seven-months-old Guernsey bull; sudden attack complete paraplegia. Destroyed six weeks later, and at that time atrophy of gluteal muscles was noted.

B. Holstein heifer, six months old. Animal eating, suddenly completely paralyzed in hind quarters. Destroyed a week later.

Dr. Mason also reports cases of this character as not uncommon in the past few years.

No. 18.

Amesbury. — Dr. A. A. Taylor. Twenty-year-old gelding became paralyzed in stable. This horse had been working every day. Destroyed. The owner of horse was G. P., Hunt's Corner, Salisbury.

No. 19.

Amesbury. — Mr. Morrison. Mr. Morrison is a dog breeder, and also the dog officer for the town. One case of paralysis (paraplegia) in a dog, with no apparent cause.

No. 20.

Amesbury. — E. S. Worthen. Dog twelve years old became suddenly paralyzed. This case seen December 2. The dog was completely paralyzed posterior to the lumbar regions. Appetite and general health excellent. The attack occurred about November 8, and when seen showed no atrophy.

No. 21.

Boston. — Dr. J. W. Tobin. November, 1910. Dog, nine years old; paraplegia. The dog became paralyzed at Braintree and was sent to Dr. Tobin's hospital. The dog was taken home by the owner, so that the case was not seen again.

No. 22.

Boston. — Dr. S. F. Wadsworth, Cummington Street. *A.* Three cases of paralysis in dogs during the last week in September and the first week in October, 1910. All 3 cases had the same symptoms: slight paralysis of hind legs, increasing steadily until paralysis of all four legs, coma and death. The head was twisted to the right. The dogs affected were of different breeds and ages. Two lived twelve days, and the third, a puppy, lived only three days.

B. A cocker spaniel on October 11 showed a slight paralysis of the hind legs. The paralysis increased rapidly, and in a few hours there was complete paraplegia. Treatment consisted of point firing in the lumbar region, with cautery. December 5 dog able to walk and improving steadily. No atrophy.

No. 23.

Gloucester. — Dr. E. W. Babson. A previous report has been made by Dr. Babson on a case seen during the winter of 1909–10.

Case of paralysis (paraplegia) occurring in a horse while being driven. The horse removed to the stable and put in slings. Improved slightly for a few days following the administration of a purgative. The paralysis later became more pronounced and the patient was destroyed.

No. 24.

Beverly. — Dr. H. D. Lambert. Chicken paralysis among his own game fowls. There were 5 or 6 cases early in September, all dying.

No. 25.

Beverly. — Dr. W. B. Wentzell. Called to attend 15 cases of paraplegia in cows during the past year. The cows affected have been brought to Beverly on the cars from without the State. The different shipping points are not known. The disease occurred during the summer months in 70 per cent. and in the early fall in 30 per cent. The paralysis appeared in from one to four days after unloading. The greater majority of the cases died.

No. 26.

Salem. — Dr. J. H. Seale. L. & S. stable, W Street. *A.* Horse sick with gastro-enteric disturbance for two days. The third day paraplegia suddenly developed, and the horse died three days later. Case occurred during summer of 1910.

B. Horse affected with colic and diarrhœa while at work; returned to stable, and paraplegia developed in a few hours. The paralysis progressed until all four limbs were affected. The paralysis was more marked in the forward legs. Died quietly five days later. Cases A and B occurred during the summer.

C. L. & S. stable, F Street. Horse purchased about November 1, fresh from the west. Developed paralysis of the hind quarters about December 1. Symptoms, swaying, staggering gait; bowels and appetite normal. Temperature variable, ranging from 100.4° to 102.4° F.

D. At the same time with cases A and B there were 8 or 10 cases of gastro-enteric disturbance among the horses in the same stable. No change in water supply or new hay or grain.

E. Several cases of paraplegia, in cats and dogs, accompanied by vomiting. The later cases terminated fatally.

No. 27.

Lynn. — Dr. F. J. Babbitt. *A.* Cow owned by F. J. G., Lynn. The cow was purchased at Brighton about Sept. 1, 1910, and two weeks later developed paraplegia. The cow was being driven from the pasture to the stable, and fell down while crossing the road, regained her feet and a short distance further fell again. Symptoms: complete paralysis of hind quarters, anorexia. The head seemed peculiarly sensitive, as the lightest touch would produce muscular spasms, twitching and finally coma. Later, consciousness regained. Died five days after onset.

B. Chicken paralysis very prevalent on the farm of F. J. G. during 1908.

C. Cow owned by R. G. Partial hemiplegia; head turned to one side (right) and cow circled to the right when forced to walk. The first symptom was the twisting of the neck, and occurred in the stable. Destroyed after three days.

D. Numerous cases in cats during the past few years. The paralysis gradually increased in severity and involved the rest of the body, resulting in death.

No. 28.

Reading. — Dr. C. S. Playdon. Six cases of paraplegia in cocker spaniels in the month of November, 1910. The dogs varied in age from four to seven years. The only difference in the cases was that some were very sensitive to the touch. The greater number made a complete recovery.

No. 29.

Newburyport. — Dr. F. C. Blakely. Cattle owned by F. S. M., Newburyport. In a herd of cattle 5 cases of eczema appeared among the young during the month of May, 1910. The eczema was followed by paraplegia in 3 cases.

A. Heifer. Eczema of abdomen, May 3, followed rapidly by eczema of neck and legs. Symptoms of paraplegia, with diarrhœa, May 25; May 26 paraplegia complete, animal down; May 27 animal able to get up, and improvement continued slowly. Case not seen for several months, but on December 6 no paralysis or atrophy.

B. Bull, four years. Eczema, May 24. Paraplegia developed May 26; bowels constipated May 26, followed by dysentery (bloody); paraplegia complete May 27. Paralysis continued, and animal died June 5.

C. Heifer. Hæmatoma in the skin on the body, one on neck and shoulder and also eczema, May 24. No paralysis or diarrhœa. Recovered.

D. Hæmatoma in bronchi, demonstrated on autopsy. Paraplegia. Paralysis increased in area and animal died.

E. Heifer. Bloody diarrhœa. Animal destroyed.

No. 30.

Boston. — Dr. A. W. May. Spaniel, Mrs. H. B. C., Jamaica Plain. This dog had a slight paraplegia for a short time, and during August, 1905, suddenly developed complete paralysis of the hind legs, bowels and bladder. The case improved slowly. The paralysis and atrophy are evident at the present time.

In a livery stable owned by Mr. C., 7 cases of paralysis occurred Sept. 30 and Oct. 1, 1910.

A. Patient was visited in the evening, September 30. The horse was in a comatose condition, respiration slow and subnormal temperature. The horse did not eat and was unable to drink on the 29th. Paralysis of the hind quarters during the morning of the 30th. The paralysis increased rapidly, until the fore legs were affected; the patient fell down, became unconscious and died in the evening.

B. Patient visited during the morning of October 1, and showed complete paralysis of the muscles of deglutition; blind, with extreme dilatation of the pupils; constipation, retention of urine; temperature 102.5°; and slight swaying of the hind quarters. Paralysis increased until animal became comatose, in the afternoon, and died late in the evening of the same day.

There were 7 cases of paralysis, including the 2 described, all having the same train of symptoms. The cases terminated fatally, with one exception, within sixty hours of the first symptom of paralysis.

In 1 case the symptoms were less severe, and the patient was supported in slings, and lived until October 20.

In making an examination of the other horses in the stable, about 15 in number, it was found that 5 were suffering from a gastro-enteric disturbance. The symptoms were colic, diarrhœa, tympanitis and anorexia. The latter cases made complete recoveries in two or three days.

The general condition of the affected horses varied from excellent to very poor. The ages were from six to twenty years. Inquiries were made as to a possible source of lead causing poisoning, but with negative results. It was found that, for several days previous to September 30 raw potatoes had been fed to the horses. The symptoms of paralysis did not appear until the potatoes had been wet, as they lay in a pile, and mold had developed. The potatoes were not fed after October 1 and no new cases appeared.

AN INVESTIGATION CONCERNING INFANTILE PARALYSIS AS IT OCCURRED IN THE CITY OF FALL RIVER IN 1910.

In the summer of 1910 there occurred in Fall River an epidemic of infantile paralysis of considerable extent. The exact number of cases occurring in this epidemic is not important for the present purpose, nor did the general study of ages, grouping dates of onset, etc., show anything of especial importance. The locality was studied by Dr. T. P. Hennelley, who was delegated for the purpose by the Board. The most important part of the investigation seems to be in the results of the study of contact of the cases, the frequency of indefinite illnesses in the same family or neighborhood, and the curious tendency of the disease to remain for years in one locality, with also a tendency to recurrence in the same family. The cases will be presented individually, with such points as seem of interest noted.

The study of the cases with regard to transmission, in a crowded district among a foreign-born population, is naturally not a matter which can be dealt with with scientific exactness, but available facts bearing on each case can be presented and reasonable inferences pointed out.

The cases to be presented seem to suggest that in most instances there was present direct or indirect contact with a positive case or a case of illness which was possibly abortive infantile paralysis.

To facilitate the study of the case, the case numbers are here grouped to indicate what the individual cases suggest from an etiological point of view.

Suggesting contact with cases of indefinite illness: —
1, 7, 11, 12, 15, 17, 21, 23, 27, 28, 29, 32, 33, 35, 37, 38, 41, 42, 49, 50, 51, 61, 62, 65, 66, 69.

Suggesting direct contact with positive case: —
18, 23, 31, 44, 52, 55.

Suggesting indirect contact with positive case: —
26, 32, 38, 41, 43, 52, 57, 70.

Suggesting transmission, direct or indirect, to another case: —
4, 10, 14, 18, 19, 29, 40.

Persistence of the disease in a given locality: —
1, 21, 25, 65.

Previous occurrence of paralysis in same family: —
3, 4, 8, 56.

Association with domestic animals possibly or definitely diseased: —
1, 2, 20, 27, 33, 49, 66, 68, 72.
Of interest for other reasons: —
6, 45, 67.

Fall River is a manufacturing city of 119,295 inhabitants and all parts of the city are in close proximity to water. The Taunton River forms its western border, the Watuppa, north and southeastern borders, while the Quequechan River almost divides the city in two by its course westward from South Watuppa Pond.

In most instances more than one family lived in the same house and all used the same entrance stairway. More than one-half of the houses were closely packed together, with little or no intervening space.

Grape vines were present in many of the yards. Some yards were grassed over; others were composed of gravel or cinders.

Only one case (28) occurred in either of the two orphan asylums, and the large majority of cases occurred in French-Canadian families.

Almost all the patients visited one of three beaches on the Taunton River, but in the vicinity of none of these beaches were cases present, except that, at Lanagan's Beach, in the extreme north end of the city (Steep Brook cases), three and possibly four cases occurred within a radius of one hundred to four hundred yards. Most of the cases occurred in those districts where children of susceptible age were most numerous and where sanitary conditions were poorest.

Various dealers in vegetables, milk, etc., come into Fall River daily. Most of these come from Westport, Somerset or Swansea.

In only one case did a milkman leaving milk in Fall River have a positive case in his family. A suspicious illness, however, occurred in the family of another milkman residing and delivering milk to Fall River. There was a child with a positive case in the next house.

Case No. 1.

Female, two and one-half years. This case is of interest in two respects. First, the child had a typical attack of the disease from May 5 to May 10, 1910, followed by loss of power in the right leg, with recovery from the paralysis in about a month. On June 28, 1910, fever, diarrhœa, etc., again came on, followed by tenderness in the legs and inability to walk. A month later the child was able to stand, but showed toe drop in the right leg and double flat foot. The case, of course, was not observed carefully enough to warrant a definite statement that two attacks occurred, but the history and examination of the case suggested it strongly.

At the time of the first attack a sister was ill with vomiting and constipation.

Secondly, the neighborhood seemed particularly liable to the disease. In the same room a case had occurred seven years before; 150 yards away a case fourteen years before; 50 yards away a case two and one-half years before; and in the immediate neighborhood two other cases had occurred two and two and one-half years before.

Six cases had thus occurred in previous years in this immediate neighborhood, as shown by the map. A cat in the family had been affected by a throat trouble for three or four years, and choked when it ate. The child had played much with this cat.

The case shows the persistence of the disease in a given locality.

Case No. 2.

Female, one and one-third years. Russian Jew. Father a dealer in skins of cows, stored in barn.

District congested, and no definite history of contact. Two other children, six and three and one-half years, not affected.

Case No. 3.

J. H., male, one year and five months. Onset, June 7, 1910; paralysis, June 11, 1910. Father a second hand in spinning room.

Child's mother has cousin whose son (now twenty years old) had anterior poliomyelitis at two years of age. No direct contact. Two deaths in same family previously.

(1) Two-year-old boy, about Thanksgiving, 1907, suddenly ill with fever, stupor, lasting one day; paralysis of one leg noted a few days later.

(2) Four-year-old boy, taken suddenly ill Jan. 28, 1908; fever, vomiting, stupor, inability to swallow. Died Jan. 31, 1908. Before death became very red in face.

Summary. — A previous case in a distant relative and two previous deaths in family, possibly from infantile paralysis.

Case No. 4.

M. H., female, two years. Onset, June 11, 1910; paralysis, June 12, 1910. Father a laborer.

Three years ago, at a different address, an older child in the family had infantile paralysis. There is no knowledge of contact with a known case, but four children in the next house (same yard) are playmates. One of these children, age one year, about two weeks after the onset of case No. 4 had an attack of diarrhœa, vomiting and fever, lasting two days. No physician was called and the child apparently recovered. On examination it was found that this child used both legs with equal strength, but there was atrophy and flatness of the outer aspect of the upper part of left thigh. Across the street from the house of case No. 4 is a barn with several hens, and with manure in the yard.

Summary. — No history of contact. Possible transmission to another child.

Case No. 5.

B. C., female, seventeen months. American French-Canadian. Onset, June 13, 1910; paralysis, June 16, 1910. Death from broncho pneumonia, due to measles, July 10, 1910.

No positive case known in vicinity. Mother, without baby, however, recently visited in infected districts on Lowell and Nashua streets. Another child died in February, 1909, of "brain fever," being possibly a case of infantile paralysis, although the season of the year makes it unlikely.

Summary. — No known contact. Possible previous case in family.

Case No. 6.

E. C., male, sixteen months. American. Onset, June 17, 1910; paralysis, June 27, 1910.

Excellent grass yard, high elevation, no traffic on street. Father drives a team collecting wet wash and soiled clothes, which come in all sorts of receptacles. His route, however, is mostly confined to a district which was little affected. In fact, at the time of onset no case was known in this district, and the father collected no laundry from a house where a positive case occurred, nor, in fact, from any house where he knew positively of any illness. He does not touch the clothes, but does handle the baskets, bags, cans, etc., coming from the houses. Flies and mosquitoes very numerous around the house and yard.

Summary. — Case with no known contact. Possible source of infection in soiled clothes.

Case No. 7.

A. L., female, twenty-two months. French-Canadian. Onset, June 17, 1910; paralysis, June 19, 1910. Father keeps grocery store. Other children, six, five and three and one-half years.

Noteworthy is the occurrence of several minor illnesses in children of the neighborhood about the same time. The family live under very good conditions. In the yard behind house is a shed which was formerly used as a barn, and which contains a privy. The privy has not been used for two years, however. The physician in this case prescribed syrup of iodide of iron, which tasted so good to the well children that they licked the spoon after the sick child had used it. This occurred several times, but no illnesses resulted in three children of the same family, six, five and three and one-half years old.

Contemporary Illnesses in Neighborhood. — (*a*) Two children with fever and threatened convulsions early in June. (*b*) Early in June baby with fever, vomiting and diarrhœa, and inability to walk; recovery in few days. (*c*) Baby with summer complaint. (*d*) Child with summer complaint, July 3.

Summary. — Possible abortive cases in neighborhood, as shown by preceding or contemporary illnesses. Failure to infect of a spoon contaminated with secretions of mouth of a patient in acute stage of disease.

Case No. 8.

D. G., male, three years eleven months. French-Canadian. Onset, June 19, 1910; paralysis, June 22, 1910. Another child of fourteen months not affected.

Father had infantile paralysis at two years, when living in Canada, and is still lame. Family lived under excellent conditions, except for numerous mosquitoes beneath the trees in the back of grass yard, and of ants in the grass. Many children in the neighborhood came in contact with this child. Mother knew of no illnesses. This patient presented on arms and legs numerous pigmented spots, representing infected mosquito bites of varying ages.

Case No. 9.

E. F., female, fifteen months. American. Onset, June 21, 1910; paralysis, June 28, 1910. Father a fish dealer.

This case occurred most probably at the home of the grandmother, where child remained while mother was working in the mill. This was one of the earliest cases in Fall River, yet this district was the least infected of any in the city.

Case No. 10.

A. K., male, four years three months. American Jew. Onset, June 21, 1910; paralysis, June 21, 1910. Father a fruit and vegetable pedler.

This was an extremely transient case, but was the first of three cases in different families. Nos. 10 and 18 were playmates, and Nos. 18 and 55 were playmates. All these cases occurred within 25 yards of each other. No other children in the same house ill. No. 10 and No. 18 ate cherries which grew in the back yard of No. 10.

Case No. 11.

J. W. W., male, four years. American. Onset, June 21, 1910; paralysis, June 28, 1910. Father a coal merchant.

This case lived with a grandparent in the north end of the city. A sister, eighteen months old, living with the mother, 400 yards away, had a combination of illnesses, starting about January, 1910, and ending in May, 1910, at which time the child's mother noticed that the child's feet turned in. The two children frequently came in contact with each other. Several children who came in contact with case No. 11 had mild illnesses before or after the onset of this case. During the onset of the disease the patient attended a party, at which several of these children were present, but no positive case of infantile paralysis followed.

Summary. — Contact with possible abortive case in sister. Other possible abortive cases before and after, in other playmates.

Case No. 12.

A. M., male, four years eight months. American. Onset, June 22, 1910; paralysis, June 23, 1910. Father a carpenter. Two other children, one two years old and one eleven days old, not affected.

A playmate of this case, living 100 yards away, had a suspicious illness two weeks before, just previous to which the two picked and ate some green huckleberries. None of the numerous (30 or 40) playmates of this case ever came down with paralysis. Some excavating had been done in front of the house within two or three months of onset. No positive case in the near neighborhood.

Summary. — Possible abortive case in playmate.

Case No. 13.

B. V., female, twenty months. French-Canadian. Onset, July 23, 1910; paralysis, July 25, 1910.

The neighborhood in which this case occurred is one of the most unsanitary in Fall River, and was probably an infected neighborhood. The case was within 150 yards of case No. 27, but no contact between the children was recorded. Communication between the families probably occurred.

Summary. — Possibly acquired from case No. 27 by indirect transmission.

Case No. 14.

H. H., male, age five years. French-Canadian. Onset, June 24, 1910; paralysis, June 25, 1910. Father a hand carder.

The younger child in this family probably had a mild attack of infantile paralysis shortly after this boy. There were also visits to a family in which there is a girl of thirteen, who, when six years old, had anterior poliomyelitis, and in the next house to whom lives a family in which a mild positive case occurred in 1910.

June 17 went to circus (horses and other animals, manure, etc.).

Case No. 15.

H. D. A., male, one year eleven months. Irish-American. Onset, June 26, 1910; paralysis, June 30, 1910. Father a bartender.

Lived near case No. 4. Two cases of gastro-enteritis in family on floor above. One had difficulty in eating and breathing.

Case No. 16.

J. A., male, two years. Irish-American. Onset July 5, 1910; paralysis July 5, 1910. Other children, 13, 11, 9, 7, and (2) years old. Father a janitor of public school.

This is the only case occurring in this vicinity.

Case No. 17.

G. G. C., female, two years. English-American. Onset, July 5, 1910; paralysis, July 9, 1910.

Patient lived under very good conditions. Played in excellent grass yard; no contact with great number of children. A few children in immediate neighborhood had indefinite illness at the same time.

Case No. 18.

R. B., male, four years nine months. French-Canadian. Onset, July 5, 1910; paralysis, July 6, 1910.

Two positive cases were connected with this case, one of which, No. 10, occurred previous to this case, and one, No. 55, occurred after. No. 18 probably contracted the disease from case No. 10. They ate cherries in common, picked in yard of No. 10. No. 18, after his illness, was in contact with No. 55, who fell ill later with infantile paralysis. Many children in the neighborhood. No other positive cases in the near neighborhood reported, except that baby of this family shortly after had fever and diarrhœa.

Apparent instance of direct contact and transmission to another child.

Case No. 19.

H. W. D., male, one year six months. American. Onset, July 5, 1910; paralysis, July 6, 1910.

This case occurred in a very good residential section of the city, living under the best of conditions. Up to two weeks previous to onset this family had been living for five weeks at a relative's house in Tiverton, in an ideal place, isolated and located on a peninsula extending into the Seconnet River. The child's most intimate companions were his sister and case No. 23, who lived in the next house, and who later fell ill with infantile paralysis. No other cases occurred in the near neighborhood previously. Case No. 23, the most intimate playmate, fell ill with anterior poliomyelitis a few days later. The older sister of No. 19 lived in the house with the younger (positive case) for four days after onset. The older child was then sent to the home of the same relative in Tiverton, R. I., and about ten days later is said to have developed a limp.

Case No. 20.

L. M., female, four months. American. Onset, July 7, 1910; paralysis, July 11, 1910.

This is the youngest case in the series, and was said to be purely breast fed. The locality is an infected one. The patient came in contact with no known case. A week before onset, however, child ate a little ice cream from a cone, bought at a little store. The three-year-old sister ate from the same cone and was not affected. A grocery store, kept by father of another case, is underneath this tenement.

Many rats were found dead at this time.

Case No. 21.

E. S., male, two years. Irish-American. Onset, July 8, 1910; paralysis, July 10, 1910. Father a janitor.

Mrs. W., now of Taunton, Mass., is said to have a child ill with infantile paralysis. The W.'s until recently lived close to the S. family in Fall River, and there was probably contact between the children. The child of a neighbor, about seven days before the onset of this case, had an attack of illness with fever, vomiting and diarrhœa, but no paralysis. Contact with this child took place at a lawn party four days before onset of the present case. Two 1909 cases were close by.

Case No. 22.

G. S., male, six and one-half years. Irish. Onset, July 19, 1910; paralysis, July 11, 1910. Father kept grocery store.

Case occurred early in July and at once fell in the hands of a so-called bone specialist, who treated it for several weeks as a dislocated shoulder. No known case in contact or in neighborhood. Patient is a private school child. A four and one-half year old sister had a slight digestive upset a few weeks before.

Case No. 23.

A. D., female, twelve years. American. Onset, July 10, 1910; paralysis, July 12, 1910.

This case is to be taken in connection with case No. 19. An illness, possibly an abortive case, occurred in a child who lived across the street, and who played with this child in the hammock on the second day of her illness, the day previous to the appearance of paralysis.

No. 23 played with case No. 19 and the sister. She had kissed the younger at the time of onset of its illness. On an indefinite day in July she ate cherries in the yard of case No. 19, and for one day was ill with vomiting, diarrhœa and fever. On July 6 she went with father and mother to Boston. It was a very hot day and on getting home patient went to bed immediately, on account of headache. Family ate ice cream cones practically every night, bought from a man passing the house.

A cousin two years ago lost the use of her legs for a year.

Associated Illnesses. — A. G. was ill with vomiting two or three days in the last few days of June. There was contact with case No. 23.

J. B., another neighbor, was ill for a week beginning July 5 with intestinal trouble. The last time in contact with case No. 23, July 2.

Case No. 24.

L. H., female, five years. French-Canadian. Onset, July 10, 1910; paralysis, July 12, 1910. Other children, 8, 6, (5), 4, and 1½ years old.

Case occurred in an unsanitary and infected district. Visited and was

visited by persons living in the immediate vicinity of two positive cases, respectively, cases Nos. 29 and 38. No other or definite source of contagion known.

Case No. 25.

E. M., male, two years two months. French-Canadian. Onset, July 12, 1910; paralysis, July 14, 1910. Parents are mill hands.

This child lived six days of each week at the home of the father's sister, where a child, case No. 44, came down with infantile paralysis almost three weeks later. This case was in an infected district, with numerous possibilities of contact.

Cases suggest either the infection of a house by No. 25 or a common source of infection for Nos. 25 and 44.

Case No. 26.

A. C., male, ten months. Portuguese. Other children, 12, 10, 6 years, and 10 months old. Onset, July 12, 1910; paralysis not seen until July 26, 1910. Father a laborer.

This case is probably to be connected with case No. 50, living in the front of house. No direct communication or contact between the two positive cases, but indirect contact by carriers is possible. Many children in yard.

Case No. 27.

O. F., male, three years. French-Canadian. Onset, July 14, 1910; paralysis, July 15, 1910. Other children, 11, 10, 9, 8, 4, (3), and 1 year old.

This case lived in a very unsanitary district, under bad conditions. Many suspicious cases of illness in the vicinity (see case No. 13). There were six other children in the family, but no others were affected.

The children of the house visited daily the family (six children), within 75 yards of case No. 13. One of these children died the same week as case No. 27. Diagnosis not known.

Dead cat found in cellar one week before onset. Had been killed by boys, but was said to be sick at the time.

Case No. 28.

I. F., female, five years. Orphan asylum. Onset, July 16, 1910.

The patient was only out of the institution for two days in six months, and then to attend the father's funeral, which took place from the next house to that in which a 1908 case occurred, and is only 25 yards from the fatal case No. 62.

The sister who teaches in the ward where this child lived was visited by case No. 32 previous to her onset. The two positive cases, however, never came in contact and are not related to each other. The child received visits, however, from mother and two adult sisters from time to time. No cases of

infantile paralysis in the institution for the past two or three years. Among children who entered the home about the time of onset of this case were two or three who had digestive upsets, and one could not walk temporarily.

Case No. 29.

A. M., female, three years. French-Canadian. Onset and paralysis, July 18, 1910. Other children, 4, (3) and 1 year old. Father a mill worker.

This case and case No. 38 live in adjacent houses, with fence between. A sister of case No. 29 was ill with headache and fever about two weeks before the onset of this case, — the illness lasting twenty-four to forty-eight hours. Examination of the sister reveals the slightest possible atrophy of the right lower leg, with very slight eversion of feet.

Contact with case No. 38 by means of the well children in the yard. Case No. 38 occurred a few days later.

Case No. 30.

H. Y., female, six years. American. Onset, July 18, 1910; paralysis, July 22, 1910.

This is a case occurring in the extreme north end of the city. The house on the left had a positive case two weeks before. The house on the right had a suspicious abortive case one month before. No. 30 frequently played with and took care of a possible abortive case.

Case No. 31.

E. D., male, four years. English. Onset and paralysis, July 19, 1910.

This case undoubtedly had contact frequently with a case of paralysis occurring about two weeks before.

Case No. 32.

I. F., female, fourteen years. French-Canadian. Onset, July 19, 1910; paralysis, July 22, 1910. Other children, (14), 11, 10, 8, 7, 5, 3, and 1 year old.

This case occurred directly across the street from case No. 41, which had its onset ten days later. Curiously enough the oldest child of a family of nine fell ill with the disease in this instance. The eight-year-old child had a probable abortive attack two weeks before. She came in contact with the children of the family of case No. 41.

This case, two days before onset, visited at St. Joseph's Asylum, and there came in contact with the teacher who taught case No. 28.

All the children of this family played with a sister of case No. 41. No contact, however, between actual patients.

Summary. — Probable abortive case in sister; contact with children from a house where a case occurred ten days later; contact with the teacher of a positive case.

Case No. 33.

H. O'N., female, seven and one-half years. American. Onset, July 20, 1910; paralysis, July 21, 1910. Other children, (7½), 6, and 2 years old. Father a letter carrier.

A suspicious illness in baby two weeks before. No positive case in neighborhood. Neighbor keeps cat hospital in house behind.

Case No. 34.

M. F., female, two years nine months. Onset, July 21, 1910; paralysis, July 24, 1910. Father a carpenter.

This case had been at a summer resort near Fall River for almost a month, except for returning to Fall River with mother every Friday for a few hours to collect rents.

No positive case in neighborhood either in Fall River or Ocean Grove.

Case No. 35.

D. P., female, two years. Irish. Onset, July 22, 1910; paralysis, July 26, 1910.

This case occurred in a probably badly infected district, as a case (No. 40) occurred 50 yards up the street a week later.

A playmate of this child, seven years of age, was ill at the same time for a few days and died. Three physicians previous to death diagnosticated the illness as cerebro-spinal meningitis. Death occurred four days after onset of illness of case No. 35.

Case No. 36.

H. L., male, twenty-eight months. French-Canadian. Onset and paralysis, July 22, 1910. Father a laborer in gas works. Two brothers and a sister are weavers in a mill. Other children in family, 12, 10, 7 years, (2 years 4 months) and 4 months old.

Case No. 37.

J. L. B., male, five years. American. Onset, July 23, 1910; paralysis, July 26, 1910. Father a belt maker.

Baby sister, two weeks before July 4, had fever for several hours, followed by convulsions. Perfect recovery.

Case lived in sparsely settled country in outskirts of city. No positive case in the vicinity. No contact with positive case, but many children in neighborhood had suspicious illnesses.

A boy, 10 years old, early in June lived in the same house. He had an attack of vomiting, diarrhœa and cough, and was ill three days. Recovered and moved to another neighborhood, where three cases occurred later, — namely, Nos. 10, 18 and 55.

Summary. — Possible infection from doubtful illness in sister and possible connection by means of house with another case of doubtful character.

Case No. 38.

E. S., female, eighteen months. French-Canadian. Onset, July 24, 1910; paralysis, July 25, 1910. Other children in family, 9, 5½, 3 years, and (18 months) old. Mother and father work in the mills. Three-year-old child had diarrhœa, vomiting and fever a short time before.

This case is probably connected with a case occurring earlier, No. 29, who lived in next yard and had same playmates. Indirect contact by means of well children in the respective families. Many children in the yard, but no other positive case.

Several suspicious abortive cases in the vicinity. Neighborhood is crowded with children and is very unsanitary.

Case No. 39.

E. M., female, five years. English. Onset and paralysis, July 25, 1910. Father a helper in mill.

The mother of this case in January, 1910, developed a cold in the head which lasted three days. She then felt well for two weeks, and then suddenly was taken with general pain, severe headache and inability to move arms or legs, — had to be carried. It was six weeks before she could walk and three months before the pain entirely disappeared.

A child, seven years, living in the next house, is a cousin of case No. 43. This child was visiting at Flint village, where she developed a febrile attack for two days, just about one week before onset of No. 39. The parents, being afraid that their child was going to die, called in the parents of No. 39 to take care of the child.

Case No. 40.

F. L., male, three and one-half years. Irish-American. Onset, July 28, 1910; paralysis, July 29, 1910. Father a laborer. Other children in the family, 13, 8, (3½) years, and 3 weeks old.

Infected district, and case No. 35 occurred in the immediate neighborhood a week previously. Contact with M. F., four years, who, until early July, lived within 40 yards, and who moved to Rhode Island, and there one week later developed infantile paralysis.

Case No. 41.

L. L., female, six years. French-Canadian. Onset, July 29, 1910; paralysis, Aug. 1, 1910. Father a weaver. Other children, 9, 9, (6) years, and 16 months old.

This case lived directly across the street from case No. 32. No contact between cases, but contact existed between the other children of both families. District is infected and unsanitary.

Several suspicious cases of illness, possibly abortive cases, occurred in

same family at about the same time. Patients played with the well children of the family of case No. 32 across the street.

Summary. — Indefinite illnesses in family and proximity to a case occurring about ten days earlier.

Case No. 42.

A. C. R., male, two years. Portuguese. Only child. Onset, July 29, 1910; paralysis, July 30, 1910. Father conducts a grocery store in adjoining house.

Seventeen-months-old child, up stairs, ill with stomach trouble about a week before. Case No. 63 occurred 40 yards away, but no known contact occurred with positive case.

Case No. 43.

N. D., male, eighteen months. French-Canadian. Onset, July 30, 1910; paralysis, July 31, 1910. Only child. Father a mill spinner.

The mother of this case was acquainted with the families of cases Nos. 39, 36, 52 and 46, but denied contact with these cases. The patient was in contact with many children, — mostly in her own yard.

The parents of case No. 43 had a relative living in next house to case No. 39, and cared for a sick child in this relative's family just previous to the attack in their own child. (See No. 39.) A girl, three years old, living down stairs in the next house, had sore throat, headache and fever one week before.

Case No. 44.

B. O., female, seven years. French-Canadian. Onset, July 31, 1910; paralysis, Aug. 3, 1910. Nine other children, 24, 23, 18, 16, 14, 12, 11, 9, (7), and 2 years old. Father a stove driller.

This case is connected with case No. 25. When case No. 25 was ill, case No. 44 visited the house.

Case No. 45.

E. F., male, three and one-half years. American. Onset, July 31, 1910; paralysis, Aug. 3, 1910. Father a letter carrier. One other child in the family, four months old.

Probable connection with case No. 47 and with a very suspicious mild case. None of this patient's intimate companions was ill.

Father delivers mail at the house of a case which occurred about July 19, the full report of which it was impossible to obtain.

An aunt of case No. 47 took care of No. 47 while the mother worked, and was visited by the mother of No. 45 frequently. The last visit was two weeks before the onset of case No. 45.

Evidence of contagion in this case is very slight, but the district is infected.

Case No. 46.

J. St. G., female, three and one-half years. French-Canadian. Onset, July 31, 1910; paralysis, Aug. 2, 1910. Father a weaver. One other child, five and one-half years old.

This case was a light one. No paralysis found at time of investigation. This case lives on floor above a physician, who reported several cases of infantile paralysis. The patient visited almost daily a relative at a house within 20 yards of the positive case No. 36.

Contact between this relative and family of case No. 36 is quite certain. The district is an infected one.

Case No. 47.

R. C., male, five and one-half years. English. Onset, Aug. 4, 1910; paralysis, Aug. 6, 1910. Other children, 18, 17, and (5½) years old. Mother works in the mill.

This case is probably connected with case No. 45. Many children came in contact with this child at Playground Park near by North Park.

Case No. 48.

M. M., female, one year. Portuguese. Onset, Aug. 4, 1910; paralysis, Aug. 5, 1910. Fatal case. Mother works in the card room of the mill. Other children, 20, 18, 16, and (1) year old.

No definite information obtained, owing to difficulty of making family understand. Rather a sparsely settled district, with many children, however. Families are all Portuguese or French-Canadians. Child lived under very unsanitary conditions.

Case No. 49.

A. V., female, three years. French-Canadian. Onset, Aug. 5, 1910; paralysis, Aug. 8, 1910. Fatal case. Father and mother cotton mill hands. Other children, 15, 11, 6, and (3) years old.

This case is to be taken in relation with a cat which died at almost the same time after an illness of a few hours.

The child was taken ill August 5 and died August 8. During the illness the cat was in bed with the patient. During the day of August 8 the cat was taken ill, became paralyzed in extremities and died at 7 P.M. The child died at 9.30 P.M. Many suspicious (abortive) cases in neighborhood, particularly on first floor of the same house.

Case No. 50.

A. St. P., male, thirteen months. French-Canadian. Onset, Aug. 5, 1910; paralysis, Aug. 8, 1910. Father a fan tender in mill. Nine other children, 23, 18, 16, 14, 12, 9, 5, 3, 2 years, and (13 months) old.

This case is possibly to be connected with case No. 26. Mother of this case said, "Many children of the neighborhood and a few of her own had measles in July, without the care of a physician."

Many possibly abortive cases in district, which is crowded and unsanitary.

Case No. 51.

A. G., male, four years. French-Canadian. Onset Aug. 7, 1910; paralysis, Aug. 8, 1910. Father a mill hand. Other children, 9, 6, (4), and 1 year old.

This case occurred at extreme end of city. No positive case within ½ mile or more. Very few houses in the immediate neighborhood. One-year-old child sick for two months, beginning one week before onset of present case with diarrhœa and fussiness, but no paralysis. Another six-year-old child in the family was in bed with fever, vomiting and delirium three weeks before onset of present case. Sick a week.

Case No. 52.

T. L., male, twenty-one months. French-Canadian. Onset and paralysis Aug. 7, 1910. Father a weaver. One other child, four months.

Infected neighborhood. Diarrhœa very common. This case visited a house in the same yard in which case No. 36 occurred on July 22, and near which case No. 46, paralyzed August 2, daily visited.

Case No. 53.

I. R., female, thirteen months. French-Canadian. Onset Aug. 14, 1910; paralysis, Aug. 21, 1910. Father a mill hand. Other children, 7, 6, 4 years and (13 months) old.

Difficult to obtain important information owing to the long delay before the case was reported, but the case occurred in an infected neighborhood.

Case No. 54.

H. T., male, two years. French-Canadian. Onset, Aug. 15, 1910; paralysis, Aug. 17, 1910. Father a coal teamster. Other children, 6, 4, and (2) years old.

This child was living in Assonet, Mass., when taken ill. Assonet lies at the extreme north end of Fall River. The mother states that she heard of two children in different houses in Assonet ill with infantile paralysis. Names and addresses not known.

No contact directly or indirectly with a known positive case.

Case No. 55.

H. S., male, two and one-half years. American. Onset, Aug. 15, 1910; paralysis, Aug. 17, 1910. Father a loom fixer. Other children, 3½ years, (2½ years), and 7 months old.

This is the last of three cases, probably connected. The other cases are Nos. 10 and 18. The chain of transmission runs probably from No. 10 to No. 18 to No. 55.

Several suspicious (abortive) cases connected with this patient.

Case No. 56.

F. S., male, two and one-half years. English. Onset, Aug. 16, 1910; paralysis, Aug. 23, 1910. Father a loom fixer. Other children, 9, 6, (4½), (2½), and 1½ years old.

This is a family having two cases occurring, one this year, 1910, and one four years ago in a different house. The old case is still wearing a brace, and is now four years and eight months old, having had infantile paralysis when eight months old. Father's brother had one leg everted and shortened since childhood; motion not limited; now over fifty years old; lives in England.

No other positive case in near vicinity, but baby of same family had spell of fever and diarrhœa in July and August.

Case No. 57.

O. P., female, four years. French-Canadian. Onset, Aug. 17, 1910; paralysis, Aug. 21, 1910. Father a carpenter. Other children, 13 and (4) years old.

This case and case No. 6 were the only two cases occurring in the proximity of the water supply of Fall River.

This case occurred in an undeveloped area less than 600 feet from North Watuppa Pond. The family's water supply was obtained directly from the pond by pail. Indirect contact with positive cases Nos. 38 and 29, through means of the children who lived in the same yard with these cases. Case also visited in houses where these cases occurred.

Indirect and possibly direct contact with two preceding cases.

Case No. 58.

E. B., male, seven years eight months. French-Canadian. Father a carpenter. Other children, 24, 21, 19, 17, 15, 13, 11, 9, (7½), and 4 years old.

No known illness in neighborhood and no evident source of contact.

Case No. 59.

L. D., female, six years. French-Canadian. Onset, Aug. 19, 1910; paralysis, Aug. 24, 1910. Father and three older children work in laundry and have much to do with handling dirty clothes. Other children, 17, 16, 14, 12, 9, 7, and (6) years old.

This case lived about 300 yards from cases Nos. 35 and 40. A questionable series of three suspicious illnesses in a family at the end of the same lot of tenements. The children of this family were in contact with patient, even during her illness with tonsilitis, which preceded the paralysis.

Case No. 60.

H. B., male, four years. American. Onset and paralysis, Aug. 20, 1910. Father drives team for yeast company. One other child, 7 years.

This case occurred about 50 yards from cases Nos. 40 and 35. Possible connection by way of healthy persons between this case and that of a child dying of infantile paralysis in Rhode Island more than a month before. A person who had been at the above funeral fondled the patient. Mother and sister had dysentery just previously.

Case No. 61.

J. R., female, twenty-one months. French-Canadian. Onset, Aug. 22, 1910; paralysis, Aug. 24, 1910. Father drives brewery team. One other child, three and one-half months old.

This case came in contact with several children having minor illnesses, and also with a chronic 1908 case. Baby sick with fever, vomiting and diarrhœa in the middle of July.

Case No. 62.

G. P., male, four years four months. French-Canadian. Onset, Aug. 25, 1910; paralysis, Aug. 26, 1910. Father a weaver. Other children, 12, 9, 7, 6, (4), 3, and 2 years old.

Contact with a child, age two, who early in August, 1910, had a three days' illness with fever and staggering gait; walked well at the end of week.

Other suspicious (abortive) cases in family of a neighbor a few days after death of this case.

Case No. 63.

W. M., male, seven months. Portuguese. Onset, Aug. 23, 1910; paralysis doubtful. Father a mill hand. Only child.

Case seen late and data not satisfactory.

Case No. 64.

E. H. B., male, seventeen years. American. Onset, Aug. 30, 1910; paralysis, Sept. 3, 1910. One other child, thirteen years.

This case, the oldest of the series, up to within two weeks of onset lived near cases Nos. 23 and 19, with whose family the patient was acquainted, but no known contact occurred. Case No. 64 on August 28 went to Block Island, R. I., where on August 30 he had his onset. The infection may thus have been acquired either in Fall River or Block Island.

Case No. 65.

R. L., male, eight years. English. Onset, Aug. 30, 1910; paralysis, Sept. 3, 1910. Father a foreman in mill. Other children, 10, (8), 4, and 2 years and 10 months old.

This case lived in neighborhood where cases in the past have occurred. (See case No. 1.)

It is said that in the adjoining house, same yard, a child was ill earlier in the summer. Later, the child could not walk; is now well.

A series of cases from Westport are reported in this connection. Westport lies just across Watuppa Pond, and is in intimate contact with the city of Fall River. The following cases occurred in Westport: —

Case No. 66.

C. D. M., male, 9 years. American. One other child, 7 months old. Onset, July 27, 1910; paralysis, July 4, 1910.

A child from Fall River was ill with vomiting and diarrhœa for a few days about two weeks before the onset of the present case and this child had been at the house where case No. 66 occurred three days before onset.

Three children living across the street from case No. 66, the only near neighbors, all had attacks of vomiting and fever about the middle of July. Their illnesses lasted only a few days. The ten year old child, however, was sicker than the others and was very weak in his legs so that one day he was found in a heap on the ground.

The father of case No. 66 is a truck gardener and also deals largely in eggs. He has over 700 hens and it is said that some of these hens limp all the time. Such hens are generally killed.

Indefinite illnesses in three neighboring children.

Case No. 67.

E. R., male, two years. English. Onset, June 30, 1910; paralysis, July 1, 1910. Other children, 18, 16, 13, 5, 3, and (2) years old. Father is a milkman delivering milk to Fall River families daily. He collected unclean milk bottles from a large number of customers, which bottles were cleaned by his wife and then returned indiscriminately to customers in Fall River. No definite connection could be made, however, between this milk supply and the disease as occurring in Fall River.

Case No. 68.

G. McC., female, seven years. American. Onset, July 22, 1910; paralysis, July 24, 1910. Only child. Father a fruit pedler.

A cat in the family had been paralyzed in hind legs for two weeks, but child did not handle it.

Case No. 69.

M. A. B., female, four years. American. Onset, July 20, 1910; paralysis, July 23, 1910. Other children, (6 years), (4 years), and 2 months old.

A sister on July 6 complained of being tired, and shortly after had a number of convulsions. July 8 the baby was ill with diarrhœa and vomiting. Diarrhœa said to be prevalent in a boys' camp 1 mile away.

Case No. 70.

L. A. S., female, nine months. American. Onset, Aug. 1, 1910; paralysis, Aug. 5, 1910. Other children, 9 years, 4 years, and (9 months) old.

All the family, except wife and mother, had measles two months ago. A nine-year-old brother was in the habit of carrying written messages to a house in which three cases of indefinite illness and one case of paralysis occurred.

Case No. 71.

E. A. W., male, three years. American.

A child from New Bedford was visiting the mother of patient. Th had had some illness, followed by a staggering gait and also by imp of speech.

Details concerning this contact lacking.

Case No. 72.

H. T., male, eight years. Irish-American. Father a miller. Oth dren, 17, 15, 13, 10 (8), 7, and 5 years old.

All the other children had attacks of vomiting, fever and diarrhœa, from two days to two weeks, all occurring since July 4. Patient was habit of playing with kittens and pigeons, and three pigeons were dead within three weeks of the onset of the patient's disease.

Case No. 73.

R. F. K., male, eight years. American. West Road. Onset, Au 1910; paralysis, Aug. 28, 1910. Children, 17, 14, 13, and (8) years old

Patient had measles six weeks before. In fact, all four children h measles recently.

No points of contact found.

Case No. 74.

A. T., female, six years. American. Onset, Aug. 29, 1910; para Sept. 1, 1910. Father drives a butcher's cart. Other children, 12, 1 5, and (6) years old.

Patient had been to Horseneck Beach, a locality associated appar with other cases of infantile paralysis.

Case No. 75.

S. O. D., Jr., male, three years. American. Onset, Aug. 2, 1910; ralysis, Aug. 5, 1910. Father is a clergyman. Other children, 6 y (3 years) and 5½ months old.

Family came from Concord to New Bedford on July 18 by train, thence to Westport by automobile.

A neighboring boy of seven years had a feverish turn, with vomiting, days later.

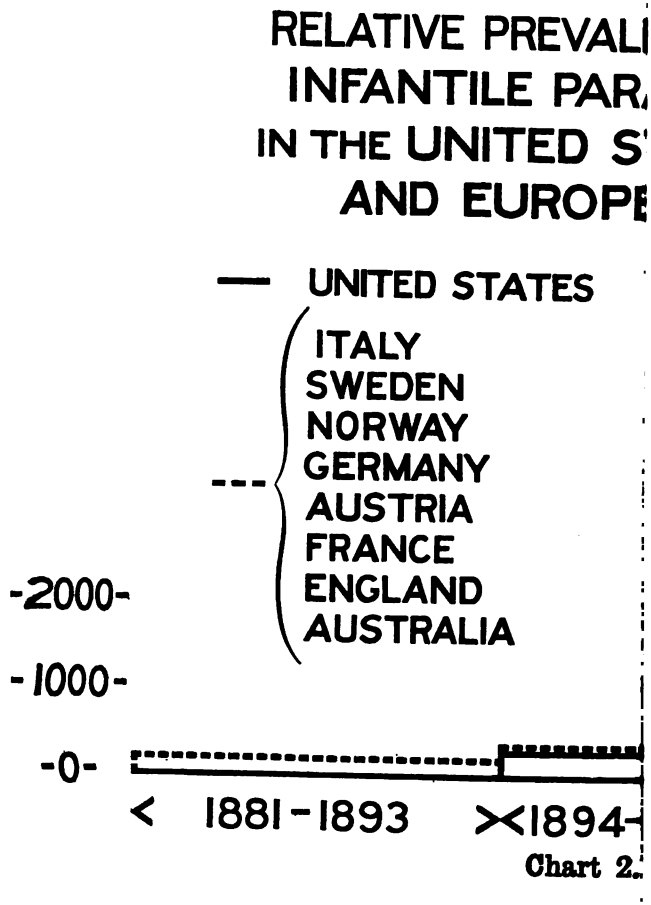

Chart 2.

The present paper [illegible]
accepted knowledge of the various aspects of the disease and an analysis of the studies in Massachusetts made for the four years beginning in 1907 by the State Board of Health.

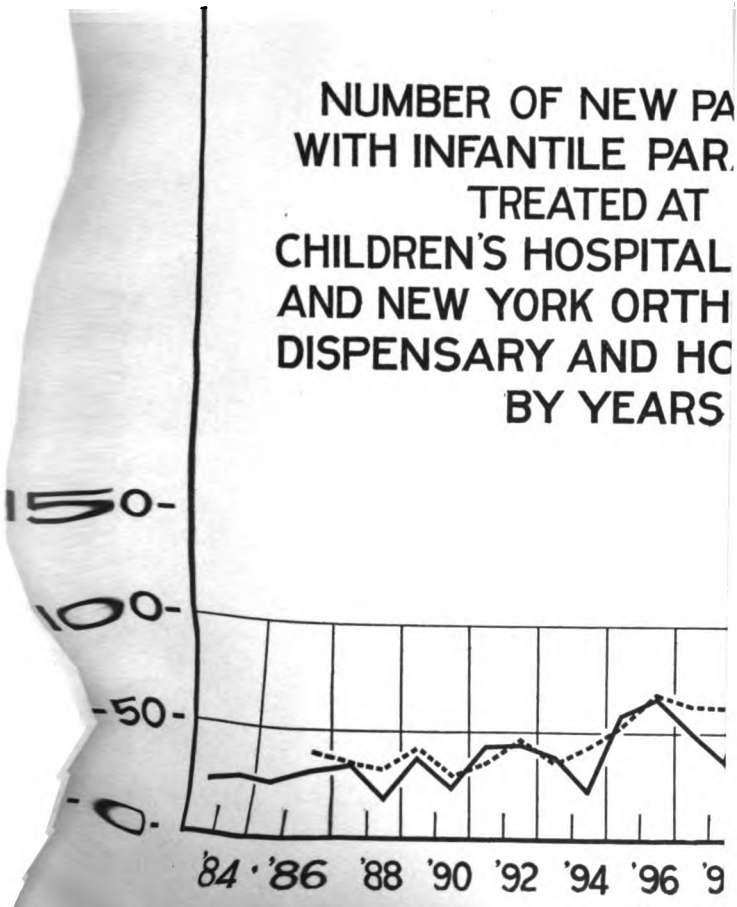

Chart 3

The present paper [illegible] the present knowledge of the various aspects of the disease and an analysis of the studies in Massachusetts made for the four years beginning in 1907 by the S[illegible] of Health.

INFANTILE PARALYSIS WITH ESPECIAL REFERENCE TO ITS OCCURRENCE IN MASSACHUSETTS, 1907-1910.

By Robert W. Lovett, M.D., of Boston, and Mark W. Richardson, M.D., of Boston.

This paper was prepared with the co-operation of Henry P. Walcott, M.D., chairman of the Massachusetts State Board of Health, and of Theobald Smith, M.D., Milton J. Rosenau, M.D., John Lovett Morse, M.D., and James H. Wright, M.D., advisory committee on infantile paralysis to the Massachusetts State Board of Health.

In the field work the Board has had for two years (1909–10) the services of Dr. Philip A. E. Sheppard of Boston, and Dr. Thomas P. Hennelly, now of Pittsfield, Mass. The epidemic in the Deerfield valley in 1908 was investigated by Dr. Herbert P. Emerson of Springfield, then State Inspector of Health for that district. Much valuable assistance has been rendered, furthermore, by all the State Inspectors of Health throughout the Commonwealth.

The problem of infantile paralysis is definitely before the physicians of the United States. In the twenty years from 1881 to 1900 there were reported in this country 200 cases; in the five-year period beginning in 1900 and ending in 1904 there were reported 100 cases; in the five years beginning in 1905 and ending in 1909, 5,400 cases, and in one — the year 1910 — approximately 9,000 cases. Such an extraordinary increase, in which Europe has in no way shared, brings before us very seriously our obligation to investigate the disease, to take account of our stock of knowledge regarding it, and to do what we properly can to quiet the panic that exists in the minds of the laity by placing before them what we know, and by asking their assistance to help us to learn more.

The present paper will consist of a brief résumé of our present accepted knowledge of the various aspects of the disease and an analysis of the studies in Massachusetts made for the four years beginning in 1907 by the State Board of Health.

I. Recent History and Etiology of Infantile Paralysis.

Résumé of Present Accepted Knowledge.

History. — The disease is not a new one. Although the first clear description of it was given in 1840 by a German, it was fairly well described by an English physician, Michael Underwood, in 1774. Since the perfectly clear description of the disease by Heine in 1840, successive additions to our knowledge have been made, the most important contributions until very recently having been by Norwegian and Swedish authors, who had unusual opportunities for the study of the disease between 1899 and 1907.

Infectious Character. — Although it had long been suspected to be an infectious disease, this fact was not absolutely proved until November, 1909, when Flexner and Lewis of the Rockefeller Institute in New York demonstrated, by inoculation of a series of monkeys with tissues from children dying of the disease, that the process was an infectious one, placing it in the category with rabies, yellow fever and foot-and-mouth disease. This discovery was undoubtedly the most important addition to our knowledge of the disease ever made.

Epidemic Character. — That the disease was infectious was suggested by the fact, recognized as early as 1881, that it occurred in epidemics. Indeed, a questionable epidemic was reported in 1841 by a Louisiana physician. From 1881 until the present time epidemics have been reported with increasing frequency, and since 1905 the size of epidemics has largely increased. We must remember, however, that of late greater attention has been paid to the disease and a larger number of cases have been recognized, so that the increase is in a measure apparent. Nevertheless, the increase has been too great to be accounted for by any theory of increased attention to the disease.

Epidemic History. — The first great epidemic of modern times occurred in Norway and Sweden in 1905, 2,000 cases being reported in Norway and Sweden together. In 1907 the first great American epidemic occurred, 2,500 cases being reported in and about New York, since which time the disease has been increasing irregularly in the United States, making a very great increase in 1910 over any previous record.

Infantile paralysis first appeared in the United States to any considerable extent in 1894, when Caverly of Rutland reported 132 cases in the Otter Creek valley, Vt. Some ten small epidemics were reported in the United States during the next twelve years, and in 1907 the largest epidemic on record, that in New York, occurred, some 2,900 cases being reported in that year. In 1908 the disease was apparently quiescent,

only 392 cases being reported in the United States. This was followed by outbreaks in 1909 in various parts of the country, and in Cuba for the first time, with a total for the year of 2,343 cases in all.

In 1910 the disease assumed much more serious proportions, and was present in 43 States. Several States had epidemics, 500 or more cases occurring in the District of Columbia, Iowa, Massachusetts, Minnesota, Indiana and Pennsylvania, and from 200 to 500 in Kansas, Maryland, New Hampshire, New York, Rhode Island, Virginia, Washington and Wisconsin.

The reasons for this marked increase in the occurrence of infantile paralysis are not clear, but certain considerations are of interest and possibly of importance.

Question of Importation. — No explanation has been given for the sudden increase of the disease in Norway and Sweden in 1905, but after that epidemic it seems not unreasonable to suppose that the disease may have been increased in this country by Scandinavian immigrants, who arrived to the number of about 42,000 in the year 1907. The epidemic of 1907 in New York may have had its origin in this way, and the disease may have been carried in a similar manner to the middle west, but the explanation is not wholly satisfactory, inasmuch as the outbreak of the disease in the west did not conform closely to the distribution of Scandinavian immigrants. The Massachusetts epidemic can be reasonably explained on the ground that it came from New York. In fact, the New York epidemic was sufficiently large to be a possible source for the outbreaks in various parts of this country.

Travel. — It has been noted in the distribution of all epidemics that they tend to spread along the line of greatest travel; certain figures were, therefore, analyzed with regard to passenger traffic in the United States.

Steam Railways. — In 1904 there were 715,400,000 passengers carried by the railroads, and in 1908 there were 890,000,000, the average number of passengers per train mile being in the first period 46, and in the second 54, that is, there were 175,000,000 more passengers carried per year in 1908 than in 1904.

Electric Railways. — The only available figures with regard to electric railway traffic are those for 1902 and 1907. In 1902 the number of passengers carried was 5,536,000,000 and in 1907 it was 9,500,000,000, an increase in round numbers of 4,000,000,000, or about 75 per cent. in five years.

Automobiles. — Another important factor as regards increased travel is the automobile, and here the only figures which it has been possible to analyze are those relating to Massachusetts. In 1904 there were

4,000 automobiles registered in Massachusetts; in 1909, 24,000; that is, in the five-year period the number of automobiles increased six times.

Conclusions from Study of Travel. — These figures show a very decided increase in travel in the United States in five years, an increase wholly out of proportion to the increase in population. It is not at all unlikely that it is one factor by which the disease has been distributed over the country; and if this conclusion is applied to the year 1894, when the Rutland epidemic occurred, the increased communication by travel becomes very much more marked by comparison.

Having considered these general aspects of the disease we now come to the closer consideration of its etiology.

Etiology. — The disease is an infectious one, caused by an ultramicroscopic virus which passes through the closest porcelain filters, its infectious character having been proved by the fact that it can be propagated in a second and many subsequent series of monkeys from tissues of a first series.

The virus is contained in the brain and spinal cord, in the mucous membrane of the nasopharynx, in infected lymph nodes, in the salivary glands, and, in the acute stage, in the cerebro-spinal fluid and the blood.

The disease may be produced experimentally in monkeys by intracerebral, intraperitoneal, subdural, intraneural, perineural and subcutaneous injections, and by implantation in the anterior chamber of the eye. The disease has been caused, also, by the introduction of the virus into the stomach and intestines. Successful inoculation has been accomplished by rubbing the virus into a scarified or even a sound nasal membrane, and two observers succeeded in producing the disease by inhalation of an emulsion containing the virus and by implantation in the trachea. All investigators agree, however, that the intracranial route is the best for inoculation.

Inoculation of horses, calves, goats, pigs, doves, sheep, rats, cats, mice, chickens, guinea pigs and dogs have proved negative, and monkeys have proved the most satisfactory animals for study. A paralysis may be experimentally produced in certain varieties of young rabbits, and may be transmitted from one generation to another of rabbits and finally back to monkeys by successive inoculations.

The virulence of a suspension is not impaired by drying (to nine days), by freezing nor by suspension in glycerine for five months, but it is killed by exposure to a temperature of 45° to 50° C. in half an hour. It is also killed by such weak disinfectants as 1 per cent. solution of peroxide of hydrogen (perhydrol of Merck), by 1 to 500 solution of permanganate of potash, and by a powder containing menthol 0.5, salol 5 and boric acid 20.

The organism is not visible with the highest powers of the microscope, and will not grow upon the culture media in general use.

The disease in monkeys is very much like that in children, but is much more fatal; in fact, so large a proportion of inoculated monkeys die that the study of the affection clinically in them is difficult. No instances as yet have been reported in which one monkey has taken the disease from another, although long-continued and intimate contact has been maintained. The incubation period in monkeys is from five to forty-six days, with an average of eight or nine days.

Immunity. — A child or animal who has once had the disease becomes apparently immune, and the blood of children and monkeys recovered from the disease has been found to render other monkeys refractory to subsequent inoculation, the specific anti-bodies persisting in the blood for three years in one case but being absent in another case eleven years after the onset.[1] This fact, of course, suggests that a preventive serum may be hoped for, but as yet the serum treatment is wholly in an experimental stage, and not yet ready for use in cases of the human disease.

Personal Transmission, Direct and Indirect. — In discussing the question of the transmission from patient to patient, the use of the technical and restricted term "contagious" will be avoided, and the term "transmissible" will be employed.

The disease may be transmitted apparently in three ways: first, by direct contact of a healthy child with a sick one; secondly, by indirect contact by means of healthy persons passing from the sick to the well; and thirdly, in certain very rare instances a child has become ill shortly after occupying a house from which an affected family had moved.

Comparative Transmissibility. — The disease is, however, apparently less transmissible than other so-called contagious diseases with which we are more familiar.

In Minnesota a very careful investigation was conducted by Hill as to the relative transmissibility of infantile paralysis, as compared with that of scarlet fever, measles, etc., in the same territory. Of persons known to have been exposed to diphtheria, scarlet fever and infantile paralysis, the percentage contracting the disease was as follows: scarlet fever, 22 per cent.; diphtheria, 17 per cent; infantile paralysis, 6 per cent. Taking the instances of these same diseases, where one case existed in a family, the number of other cases occurring in the same family was as follows: scarlet fever, 40 per cent.; typhoid fever, 30 per cent.; diphtheria, 29 per cent.; infantile paralysis, 17 per cent. The disease is thus apparently less transmissible than scarlet fever, typhoid fever or diphtheria.

[1] Netter & Levaditi: Comptes rend. de la Soc. de Biol., 1910, 68, 855.

The same evidences of mild transmissibility were shown in an investigation by Emerson of an epidemic in the Deerfield valley in Massachusetts in 1908, where a rural community offered excellent opportunities for study of contact. Furthermore, such instances as the following are difficult to explain by any theory of personal contact, direct or indirect: —

In a sporting camp in a northern forest the five-year-old son of the proprietor had been in camp since early spring. He had never left the camp during the summer. The camp was 5 miles from any other camp or house and 10 miles from the frontier town. There had been no other children in camp during the summer and no illness of any kind in the few guests or among animals. The few guests who were there came from the large cities, put on their hunting and fishing clothes and lived out of doors. In October the child developed a typical infantile paralysis which still persists in one leg.

Evidence of transmission of the disease by personal contact from other epidemics is to be found in Wickman's study, Schidler's report,[1] an epidemic observed by Frost, and the Massachusetts studies.

Possible Factors Other than Personal Transmission. — We come next to the consideration of the possibility of fruit, dust, insects and animal paralysis as factors in the causation of infantile paralysis.

Fruit. — As to fruit, the high cost of living has undoubtedly led to a diminished consumption of meat, and presumably to an increased consumption of fruit. Traffic in fruit has increased steadily between 1900 and 1908, the increase being from one to two million tons a year, up to 1905, since when there has been no such sharp increase. So far as these figures go, therefore, it would not seem that any very striking factor was to be found in this analysis. Common observation would, however, seem to show that in Massachusetts, at least, there are more fruit stores and apparently more fruit in use than was the case five years ago.

Dust. — In order to determine the role of dust in the etiology of infantile paralysis, the rainfall of the country was analyzed for two periods: first, the five years from 1900 to 1904; and secondly, the five-year period from 1905 to 1909, when we had the greater prevalence of the disease. The total rainfall during the last five years on the Atlantic seaboard has shown everywhere a diminution, reaching a high figure in New England. On the contrary, the regions in the middle west affected by the epidemic of infantile paralysis in 1909 showed an increased rainfall during the last five years, the rainfall being as much in excess of the normal as in New England it was deficient. In the middle Atlantic States, however, including New York, the rainfall in the two periods was practically the same. Excessive dryness in the last

[1] Schidler Pediatrics, XXII. 1910, 539; Frost, Public Health Bulletin 44.

five-year period cannot, therefore, be put down as a condition pertaining to the affected regions, if one considers New England along with Nebraska and Minnesota. But we must remember that the germ is not injured by freezing or drying, and that the season of occurrence is certainly the season of greatest occurrence of dust. Moreover, a German observer found the disease in an unduly large proportion of shoemakers' children, who were much in the shops where the dried dust was flying about, and the distribution of the disease along lines of travel suggest the possibility of such a mode of transmission.

Insects. — There are certain facts connected with the disease which suggest the possibility of its being transmitted by insects. This matter will be dealt with in speaking of the Massachusetts studies.

It has been shown experimentally by Flexner that if flies are allowed to feed on the cord of a monkey dying of the disease they may carry the virus for forty-eight hours.[1]

Domestic Animals. — With regard to paralysis in the domestic animals, cases of paralysis strikingly suggestive are reported in horses, pigs, cats, dogs, rabbits, chickens and other animals. A study of the distribution of animal paralysis in Massachusetts, by means of letters addressed to veterinarians and animal inspectors, showed, however, in 1909, that the distribution of cases of animal paralysis so reported did not in any way correspond to the distribution of the human disease for the same year; but much more extensive investigation along this line is necessary.

The conclusion must be that, in a consideration of etiology, infantile paralysis cannot be dismissed as a disease due only to transmission from patient to patient, but that these other factors must also be considered as possibly important.

Season of Occurrence. — In considering the etiology of the disease one is struck with the remarkably uniform seasonal occurrence which prevails in the north or in the tropics, in Europe or America. The disease begins to appear in the early summer, reaches its height generally in July, August or September, and diminishes through October, November and December, nearly to disappear in late winter and spring. In the southern hemisphere this behavior is reversed, and the disease prevails most there in February and March, which is, of course, late summer and early fall in that locality.

Age. — The age of selection of the disease is between two and three. Adult cases are comparatively uncommon, and occur chiefly in young adults. Dividing the ages into five-year periods, the following table was constructed by Frost: —

[1] Journ. Am. Med. Assn., June 10, 1910.

TABLE 1. — *Age Incidence in Five-year Periods, expressed in Percentages.*

AGE.	New York Commission (729 Cases; Per Cent. of Total).	Lovett, Mass. (615 Cases; Per Cent. of Total).	Hill, Minn. (325 Cases; Per Cent. of Total).
Under 1 year,	8.50	7.2	6.5
1 to 5 years,	82.00	64.5	48.6
6 to 10 years,	6.40	15.9	23.7
11 to 15 years,	1.90	5.0	7.7
16 to 20 years,	.68	2.4	6.5
Over 20 years,	.40	5.0	7.0

General Incidence. — It affects all classes, rich and poor, in the cities and the country, in the tenements and in detached houses, on the top floor of houses, on the ground floor and in basements.

II. PATHOLOGY, SYMPTOMS, DIAGNOSIS, PROGNOSIS, PREVENTION AND TREATMENT.

General Grouping of Symptoms and Types.

As a working hypothesis we may keep in mind the following as to symptoms and types of paralysis: —

The infection enters the body by channels as yet unknown. In some instances this infective agent causes death from general paralysis, due to extensive destruction of certain spinal and cerebral centers. In a second class of cases this agent causes some permanent loss of power in part or all of one or more limbs, the back, the abdomen, or in groups of muscles supplied by cranial nerves. In a third class the same agent causes a very local yet probably permanent paralysis in one small muscle or small group of muscles, *e.g.,* the extensor muscles of one little finger or certain muscles of the spine. In a fourth class, weakness or transitory paralysis ending in recovery occurs as the result of the same infection. And, lastly, in a fifth class the infective agent causes fever, prostration and perhaps pain, but does not cause sufficient destruction to bring about any paralysis or weakness.

If we recognize this descending scale in the severity of the disease as depending probably either on the dose of the poison received or on the resistance of the individual, and recognize that the involvement of the spinal centers is perhaps, after all, incidental rather than essential, we may well believe that the disease is far more prevalent than is generally supposed, and especially in its abortive forms.

Pathological Anatomy and Histalogy of Poliomyelitis.

The characteristic lesions of the disease are found in the spinal cord, and are most marked in the anterior horns of the gray matter of the cervical and lumbar enlargements.

In the gray matter in the early stages of the lesions are (1) Congestion of blood vessels. (2) Œdematous softening, sometimes accompanied by hemorrhage. (3) Infiltration with cells. (The cells diffusely infiltrate as well as aggregate together in foci and about the blood vessels; they are of various kinds, but are chiefly lymphocytes and larger cells whose origin is not generally agreed upon and which are sometimes phagocytic.) (4) Degeneration and diminution in the number of nerve cells. (The cytoplasm of the nerve cell becomes hyaline and disappears, leaving the nucleus naked and necrotic, and it also eventually disappears. The degenerated cell may be surrounded and invaded by the infiltrating cells. All of the nerve cells in a given area do not always show degenerative changes, and more of them may be involved in one side of the cord than in the other. In the later stages of the disease, in extensively affected regions, nerve cells and their processes are absent, the tissue is disintegrated and there is infiltration with phagocytic cells, plasma cells, proliferated glia-cells, and connective tissue cells. Eventually, neuroglia with some connective tissue replaces the disorganized tissue, and the part may appear atrophied to the naked eye.)

In the white matter, in the early stages of the disease, the lesions consist of congestion, œdema and cellular infiltration of the adventitial lymph spaces of the blood vessels, and, to a slight extent, of the white matter as well. The infiltrating cells are chiefly lymphocyte-like cells. Small foci of these cells have been noted in the white matter. In some cases acute degenerative changes have been observed in the anterior roots.

The pia mater also shows some infiltration, chiefly with lymphocytes, which is greatest in the anterior portions of the cord in the lumbar and sacral regions.

In the spinal ganglia there may be the same infiltration as in the white matter, without marked changes in the ganglion cells.

In the later stages of the disease there is degeneration and atrophy of nerve fiber tracts, anterior roots, motor nerves and muscles, depending in extent and location upon the nerve cells which have been destroyed.

In the medulla and pons similar lesions to those in the cord are found, but the degeneration of the ganglion cells is not so extensive as in the cord.

The brain, as a rule, shows comparatively slight lesions. Congestion of blood vessels, small hemorrhages, cellular infiltration of the vessel

sheaths and of the brain substance in foci have been observed in various situations. In old cases foci of sclerosis have been observed.

In acute cases, the thoracic and abdominal organs show changes which may be observed in other acute infectious diseases. A hyperplasia of the lymph-adenoid tissue of the intestine, mesentery and spleen is commonly observed in addition to acute degeneration of the myocardium, liver and kidneys, and there may be broncho-pneumonia.

There is nothing in the pathological anatomy and histology of the disease which shows the mode of entrance of the infectious agent into the body, nor whether it reaches the central nervous system by the blood or by the lymph stream.

Symptomatology of Infantile Paralysis.

It is impossible in the time at our disposal to give more than the most cursory description of the symptomatology of this disease. If we think of it as a poliomyeloencephalitis with meningeal complications and appreciate the fact that the disease may affect any or all parts of the gray matter of the central nervous system and in all possible combinations, it is easier to understand the innumerable manifestations of the disease, and it is less likely to be overlooked and mistaken for other conditions. The best classification of the various manifestations of the disease is that of Wickman which is as follows: —

1. Ordinary spinal paralysis; anterior poliomyelitis.
2. Progressive paralysis, usually ascending, less often descending; Landry's paralysis.
3. Bulbar paralysis; polioencephalitis of pons.
4. Acute encephalitis; giving spastic monoplegia or hemiplegia.
5. Ataxic type.
6. Meningitic type.
7. Polyneuritic; multiple neuritic type.
8. Abortive type.

It will be well, perhaps, before taking up the special clinical types of this disease, to consider briefly its initial symptoms and the possibilities of making a diagnosis before the appearance of the paralysis. There is, in the vast majority of cases, nothing characteristic about the onset. The symptoms are, in general, those of an acute infection. In many instances, however, gastrointestinal symptoms predominate, while in others those referable to the respiratory tract are the most marked. Sweating, marked nervous irritability and general hyperæsthesia are present in many instances before the onset of the paralysis, but they are not at all constant. Their presence points strongly toward this disease, but their absence does not count against it. It is safe to say, therefore, that

at present there is nothing about the early symptoms to justify a positive diagnosis of infantile paralysis before the onset of the paralysis, although the appearance of the symptoms of an acute infection in a child known to have been exposed to this disease, or during an epidemic, especially if accompanied by sweating, nervous irritability and general hyperæsthesia, are sufficient to justify a probable diagnosis.

A leucopenia has been found in animals during the early part of the acute stage. It is, however, not a constant symptom in man, and is often replaced by a hyperleucocytosis. It is possible that there is a lymphocytosis in the early stages, but the evidence at present is not sufficient to show whether it is at all constant. It is possible that both the absolute and differential count of the leucocytes may prove useful in the early diagnosis of infantile paralysis. At present, however, they are of little or no assistance.

Experimentally, there is an increase in the number of cells in the cerebro-spinal fluid during the prodromal stage, the polynuclear cells at this stage outnumbering the mononuclear. After the appearance of the paralysis, however, the mononuclear cells quickly exceed the polynuclear. A fibrin clot is also often formed during the prodromal or early part of the acute stage. The cerebro-spinal fluid, in the only reported instance in which it has been examined in man during the preparalytic stage, was slightly opalescent, gave a marked protein reaction with Noguchi's test and showed an excess of polynuclear cells. The formula had begun to change to the lymphocytic, however, before the appearance of the paralysis. It seems safe to say, therefore, that in most instances a positive diagnosis of infantile paralysis can be made in the preparalytic stage by means of lumbar puncture, and that this is the only way in which a positive diagnosis can be made at this time. The value of this procedure is considerably limited, however, by the variability of the early symptoms and the consequent lack of anything to definitely suggest its use. In order to recognize the disease early, therefore, before the onset of the paralysis, it would be necessary to do a lumbar puncture on every sick child, which hardly seems a rational procedure.

Abortive Type. — What has been said as to the symptomatology and diagnosis of the preparalytic stage of infantile paralysis applies equally well to the abortive type, meaning by this term the type in which there is never any paralysis. There has been some doubt as to the existence of this type in the past, because there was no way of positively proving that the cases supposed to be of this type really were examples of this disease at all, the evidence being merely presumptive. Flexner and Clark and Frost have, however, recently demonstrated the presence of specific immune bodies in the blood of persons supposed to have had

abortive attacks of infantile paralysis. The blood serum of these patients inactivated the virus of the disease, so that the infection of monkeys was impossible. There is no doubt, therefore, that abortive cases do occur. It is probable that they are as common as those with paralysis, if not more so. The importance of the recognition of these cases, if the present conception as to the role of "carriers" in the spread of the disease is correct, is obvious.

Ordinary Spinal Type. — It hardly seems necessary to take up in detail the symptomatology of this type, with which every one is familiar. The paralysis reaches its maximum in from a few hours to three or four days, is followed by a stationary period of from one to four weeks, which is in turn followed by a period of improvement, lasting from six months to a year, the final paralysis always being less than the initial. It is impossible to prophesy in the acute stage how extensive the final paralysis will be. In general, however, the final paralysis varies directly with the initial. The final paralysis is always flaccid; its distribution usually monoplegic or hemiplegic. In most instances some of the muscles of the paralyzed extremity escape; not infrequently only one or two are involved; the reflexes are diminished or absent, sensation is only exceptionally impaired. Deformities develop as the result of contractures of the non-paralyzed muscles.

It is not very uncommon for other muscles than those of the extremities to be involved, while these escape. Such muscles are most often the erectors of the spine and the abdominal muscles. The involvement is, moreover, not infrequently unilateral. The extensor muscles of the neck, those of one side of the spine or of one side of the abdomen may, for example, be alone involved. The deformities resulting from the paralysis or paresis of these muscles are often misunderstood and attributed to other causes.

Progressive Type. — In this type the paralysis usually appears first in the legs, and gradually extends upward. In rare instances it appears first in the arms, extends downward and finally upward to the muscles supplied by the medulla. It is probable that the great majority of the cases that have in the past been described under the term of "Landry's paralysis" really were examples of this type of infantile paralysis. When the paralysis reaches the external muscles of respiration, as it very often does, death is practically inevitable, and usually occurs on the third or fourth day. The diaphragm is also sometimes involved. Death occurs sooner, of course, when this happens. This form must not be confused with that in which death results from respiratory paralysis due to the involvement of the centers of respiration in the medulla.

The Bulbar Type. — Cases of this type have in the past been usually designated as polioencephalitis superior or inferior, according to which of the cranial nerve nuclei were involved. The nuclei may be affected singly or in all possible combinations. The symptoms depend, of course, on the nuclei involved. The facial and abducens nerves are perhaps the most often affected. There may be paralysis of deglutition and of the muscles of the larynx. When the vagus is involved there are disturbances of respiration and of the cardiac action. In such instances the respiration is often of the Cheyne-Stokes type, and the prognosis is practically hopeless. Involvement of one or more of the cranial nerve nuclei is not very uncommon in connection with the ordinary spinal type of infantile paralysis. When this happens the resulting picture is a combination of the two types.

Acute Encephalitic Type. — This type was described by Strümpell many years ago under the term of "acute encephalitis of children," but it has only been recently recognized as a variety of the disease under consideration. The symptoms resemble those of acute meningitis, the deep reflexes are, as a rule, exaggerated, and the paralysis is spastic. The diagnosis is usually impossible without lumbar puncture. The prognosis as to life is better than it would appear, and that as to recovery from the paralysis much better than in paralysis due to other cerebral diseases.

The Ataxic Type. — Ataxia is a prominent symptom in a certain number of cases. In a few it is the only nervous symptom; in others, it is associated with paralysis of the cranial nerves and sometimes with a small amount of spinal paralysis. The ataxia is often distinctly of the cerebellar type.

The Meningitic Type. — Symptoms of meningeal irritation are not at all uncommon in the early stage of all types of infantile paralysis. These are so marked in many instances that they present the typical picture of meningitis. Headache, rigidity of the neck and back, vomiting, tonic and clonic spasms, strabismus, Kernig's sign, delirium, coma and other signs of meningeal irritation may be present in any and all combinations. In such cases the diagnosis on the symptomatology alone is impossible before the appearance of the flaccid paralysis. Even then it is open to doubt, because flaccid paralysis is not very uncommon in meningitis, especially if it is of the tubercular type.

A positive diagnosis can only be made in most instances by lumbar puncture. The characteristics of the cerebro-spinal fluid during the preparalytic stage have already been mentioned. During the acute stage it is clear, not infrequently under somewhat increased pressure, often

forms a fibrin clot on standing and always contains an excess of protein and of cells. The cells are chiefly of the mononuclear type, most of them being lymphocytes. These characteristics are, however, the same as those found in tubercular meningitis, the type of meningitis with which infantile paralysis is most likely to be confused. A positive diagnosis between them can only be made, therefore, by the examination of the cerebro-spinal fluid for tubercle bacilli. The characteristics of the cerebro-spinal fluid in meningococcal, influenzal and pneumococcal meningitis are so different from those in infantile paralysis that no confusion with these conditions is possible.

The Polyneuritic Type. — Pain is a prominent symptom in many instances. It is sometimes located in the joints, but is more often along nerve trunks or indefinite in its distribution. It is usually most marked in the paralyzed parts. There is occasionally tenderness over the nerve trunks. The pain and tenderness are sometimes marked enough to cause the paralysis to be entirely overlooked, and a diagnosis of rheumatism of scurvy made.

The extremities are often held rigidly, and all motion as vigorously resisted as possible because of the pain on motion. This combination of rigidity and resistance is possible, of course, only when the muscles are partially paralyzed or when some of them are intact. The failure to appreciate the significance of this combination of flaccidity and spasticity not infrequently leads to errors in diagnosis during the acute stage.

Prevention. — The question of the prevention of the disease is of great importance, and you are probably asked as frequently as we are in Massachusetts, "What shall I do to protect my children from the disease?" a question which we must answer fairly with due regard to the interests of the patient, remembering, however, that advice too radical will increase the panic which is sure to appear in a locality where the disease is prevalent. It seems proper to advise people with children that, other things being equal, it is wise not to go for the summer to a locality where the disease is known to be prevalent. If, however, people find themselves in such a locality, the children and members of the family should keep away from other children who are in the acute stage of paralysis and from children who are sick with other indefinite ailments.

In such localities it would seem wise to keep children away from children's entertainments, circuses and electric cars, and to prohibit playing with sick or paralyzed domestic animals.

Length of Quarantine. — We have at present no data as to how long the infectious period of the acute stage lasts, and any attempt to determine the length of quarantine must be mere guesswork.

With regard to the necessary length of quarantine, general consent has placed it at from two to four weeks. The recent experiments of Osgood and Lucas in this matter are, however, important inasmuch as they found an active virus in the nasopharyngeal mucous membrane of a paralyzed monkey five and one half months after the active stage of the disease had passed. The virus disappears from the nervous system in three or four weeks or less, according to Flexner's experiments.

Mortality. — The death-rate is about 7 or 8 per 100, as the disease has existed in Massachusetts, but no foreign statistics show as low a figure as this, being from 10 to 22 per cent. In persons older than ten, the mortality rate is higher than between one and ten, and under one year the disease seems more fatal.

The mortality table is interesting as showing the difference between foreign and American statistics: —

TABLE 2. — *Comparison of Foreign and American Death-rates.*

	Year.	Cases.	Deaths.	Mortality (Per Cent.).
Caverly, Vt.,	1894	132	18	14.5
Wickman, Sweden,	1905	868	145	16.7
Leegaard, Norway,	1905	577	84	14.5
Zappert, Austria,	1908	266	29	10.8
Lindner and Mally, Austria,	1908	71	16	22.5
Fürntratt, Steiermark,	1908	433	57	13.1
Krause, Germany,	1909	633	78	12.3
Müller, Germany,	1909	100	16	16.0
Peiper, Germany,	1909	51	6	11.7
Eichelberg, Germany,	1909	34	7	20.6
Massachusetts, U. S. A.,	1907-1910	1599	125	7.9

The mortality of cases in older children and in adults is higher than in other classes.

TABLE 3. — *Showing Higher Mortality in the More Advanced Ages.*

	Age.	Per Cent.
Wickman, Sweden,	12 to 32 years.	27.6
Leegaard, Norway,	15 to 30 years.	25.8
Fürntratt, Steiermark,	Over 15 years.	25.5
Lindner and Mally, Austria,	Over 11 years.	50.0
Massachusetts, 1910, U. S. A.,	Over 10 years.	20.0

Prognosis. — A few years ago it was supposed by most persons that practically all cases once paralyzed were to some extent paralyzed for life; but more careful study has shown us a better and more encouraging state of affairs. In the cases in Massachusetts in 1909, a group of 150 occurring near Boston was studied in detail, and it was reported that 25, or 16.7 per cent., had wholly recovered inside of three months. This statement was not believed, and the investigators were sent back to review their work. Each one of the children was stripped, and the movements of ankle, knee, hip, shoulder, elbow, wrist and spine were examined. The statement can be definitely made, therefore, that, of this group, 16 per cent. had wholly recovered within three months.

The commonest type of final paralysis is an affection of one leg, varying in degree from a slight limp in walking to a condition which requires the use of a brace; if both legs are involved crutches are occasionally also required. In short, a disability from infantile paralysis so great as to prevent an adult from earning his or her own living occurs in a very small number of cases properly treated, but the ultimate condition of neglected cases is frequently distressing.

In connection with the question of treatment it is important to consider the reasons for this tendency to recovery which are to be found in the pathology of the disease.

At the height of the disease there is an extensive perivascular infiltration of the blood vessels of the cord, diminishing their lumen and inducing anemia and œdema of the regions of the cord supplied by them. In addition to which there is also inflammatory œdema of the nerve tracts in the white matter, and œdema of the nerve cells in addition to any destructive process which may be going on in neighboring cells. These changes, of course, cause loss of function in the parts affected, expressed as a temporary paralysis which passes away as the perivascular infiltration subsides. This apparently accounts for the improvement of paralysis in the early stage of the disease which exists in all non-fatal cases.

But the spontaneous improvement in the paralysis of the early history of the disease is no more important than the improvement to be obtained by proper treatment in the chronic stage of the disease, after there is every reason to believe that the temporary perivascular infiltration has subsided, and the reason for this later improvement apparently rests on another pathological basis. Assuming that the phenomena just described have ended, but that permanent destruction of certain motor centers in the spine has occurred, leaving a definite motor paralysis, the question arises, what reason is there to look for improvement in this. The answer apparently is as follows: —

The spinal motor centers are grouped in cigar-shaped bundles, whose length lies in the long axis of the spine. At each level of the spine are several of these bundles which overlap each other and communicate freely. Each bundle furnishes impulses to more than one muscle, and each muscle receives impulses from more than one bundle. The blood supply of the spine comes in horizontally, and the lesions of this disease are most marked around the vessels; the distribution of the destructive process is therefore likely to be horizontal, while the distribution of the motor bundles is longitudinal. For this reason it is likely that if the destruction has not been too extensive certain parts of the motor bundles will have escaped, leaving certain cells in some of them capable of function, but which, in order to perform function, must acquire new connections and new groupings.

For this reason it will be seen, as will be maintained later on the ground of practical experience, that muscle training offers the best hope of the restoration of power to affected muscles in the chronic stage of the paralysis. It is an attempt to bring motor impulses from the brain down to the affected centers in the cord, there to establish new connections and new routes to the paralyzed muscles. The more impulses of this sort, however feeble they may be at first, to reach the affected muscles, the better the chance of establishing finally routes which may serve for the transmission of impulses strong enough to secure efficient muscular contraction.

Treatment, Acute Stage. — In the matter of treatment much progress has been made in the last four years. In the acute stage meddlesome and useless treatment by stimulating drugs and counter-irritants, has become largely discredited. Rest is very important, although cathartics and hexamethylenamin are of probable value, the latter drug being used with the object of setting free formalin in the cerebro-spinal fluid. Indeed, it has been shown experimentally that if hexamethylenamin is administered by mouth to monkeys, and if such animals are subsequently inoculated, either subdurally or by the intracerebral route, paralysis is, in a certain proportion of cases, delayed or prevented. The value of the drug, therefore, in the prevention of infection would seem to be indicated, but there is as yet no experimental evidence to show that the drug is of much use after the disease has once begun.

In view of the pathological condition, it is simply common sense to keep the patient recumbent and quiet, and to avoid irritation of the muscles supplied by the affected nerve centers. Massage and electricity should not, therefore, be used, until there is reason to believe that subsidence of the inflammation has taken place. We may probably interpret

the tenderness of the limbs as showing, while it lasts, the persistence of some degree of spinal or neural inflammation, and until that has wholly disappeared we are not justified in the use of passive movements, massage or electricity.

Some men of large experience discourage all active treatment before the expiration of six weeks at least, but excellent results are frequently obtained by beginning treatment earlier on cases characterized by slight and transient tenderness. We should, however, discourage all active treatment until tenderness has wholly disappeared.

Another point of primary importance in early treatment is the prevention of contractures and deformities due to unopposed muscular action. Except in paralysis of the back and shoulder these contractures and deformities are always preventable, the contractures of the Achillis tendo, for example, which is so frequent and so troublesome, being easily prevented by the use of splints supporting the foot at a right angle.

We read much concerning the relative merits of electricity and massage in the restoration of muscular power, but who shall pronounce as to the value of either in an affection in which paralysis varies so much in extent and severity, and the recovery from which is so surprisingly different, irrespective of methods of treatment? Both methods have as an object the stimulation of muscular fibers and the maintenance of muscular nutrition, but both are inferior to muscular training in active treatment. This method of treatment is most useful in connection with massage or electricity, and aims to cultivate the transmission of a voluntary motor impulse from the brain, down the cord to the paralyzed, weakened or stretched muscles. A flicker of motion in an affected muscle is often the forerunner of a return of some degree of power, and assistance by the hand in performing voluntary movements of the foot, for example, is often of great use.

Apparatus. — As to the use of apparatus, — when a child in attempting to stand cannot, for example, hold the knee extended in weight-bearing, or when he stands in a position of deformity, for instance, with flat foot, apparatus should be applied to restore normal relations. Nothing but harm can result from sitting or going about with a curved spine, a hyperextended knee or a foot rolled over upon its inner or outer border. Stretching of muscles and ligaments, and permanent bony deformities which are sure to result, are worse than the use of braces, objectionable as the latter are.

Contractures which exist in neglected cases are not extremely resistant early in their history, and must be removed before satisfactory mechanical treatment can be carried out. Most of them can be removed if

stretched by means of bed traction, or by the use of a series of correcting plaster bandages, or by cutting. Operative treatment, except for the minor tenotomies, should not be undertaken for two years after the attack, and preferably not then in children under ten.

Surgical Treatment, Operation. — Tendon transplantation is a most useful measure, and now that it has passed through its period of extravagant claims and unjustified condemnation, it may be said, in properly selected cases and with proper technique, to produce generally satisfactory and often brilliant results.

It consists in transplanting the distal end of a healthy muscle to the periosteal insertion of a paralyzed one, the foot or knee or arm being thus rebalanced. Since we have learned to prolong tendons by silk the range and application of the operation have increased. We plan our operation, dissect and cut off our good muscle, and if it is too short lengthen it at its distal end by heavy silk; we then insert it where we like, a new and sound tendon forming around the silk. In this way peroneals may replace tibials at the ankle, or *vice versa,* hamstrings may be carried forward and inserted into the tibia to serve as knee extensors when the quadriceps is paralyzed, and the trapezius may be substituted at the shoulder for a paralyzed deltoid.

Arthrodesis. — As a means of stiffening and making stable useless joints, arthrodesis has been found a useful measure. This operation consists in opening such joints and removing enough cartilage to secure ankylosis, and is applicable to the hip, ankle, elbow and shoulder in children over twelve, and to the knee in adults, but not to the knee in growing children.

Silk Ligaments. — For the correction of foot drop in paralyzed legs use has been made more and more of anterior silk ligaments, the idea being to prevent plantar flexion of the foot, but still to allow dorsal flexion. Heavy strands of silk are sewed into the anterior surface of the tibia, carried down under the annular ligament of the ankle and sewed into the dorsal surface of one of the tarsal bones. This operation is distinctly preferable to arthrodesis.

Nerve transplantation has not yet emerged from its experimental stage.

Finally, one of the most important things that orthopedic surgeons have learned of late years is that hope for improvement in these cases through efficient and suitable treatment need not be given up in six months or one year or ten years, but that the possibility of improvement seems to persist almost indefinitely.

III. Infantile Paralysis as observed in Massachusetts, 1907–10.

The investigation of infantile paralysis in the State of Massachusetts by the State Board of Health for the four years 1907 to 1910, inclusive, shows that during that period 2,138 cases of the disease were reported to the office of the State Board of Health. These cases were distributed by years as follows: —

1907,	234
1908,	136
1909,	923
1910,	845

It is apparent from these figures that the disease in Massachusetts has in a general way during the period mentioned increased in frequency, and it has been shown that a larger number of towns were infected each year. (For maps showing incidence of the disease in 1907–10 see page 55.)

Two-year Periodicity. — There is, moreover, in these figures a suggestion of what may be an important phenomenon, and that is the appearance of a two-year periodicity for this disease. This phenomenon is noticeable in the distribution in general. In 1905 occurred the great Scandinavian epidemic. In 1906 there was no important epidemic. In 1907 occurred the New York epidemic. In 1908 there were only a few small epidemics, and none of importance reported in Europe. In 1909 occurred the Massachusetts, Minnesota and Nebraska epidemics, two large and one small German epidemics, a large outbreak in Austria and the epidemic in Cuba. In 1910 from Europe no large epidemics have been reported so far as we have been able to learn. The occurrence of the disease in this country in 1910 has already been dealt with.

This two-year periodicity has been noted a number of times in our investigation. For instance, in 1907 the western part of the State was quite sharply affected with infantile paralysis, whereas in 1908 the disease was almost non-existent in that same region. Again, in 1909 the disease cropped up, but in 1910 hardly any evidence of infantile paralysis was noted in that neighborhood. In 1908, furthermore, in the Turners Falls district, etc., a very marked epidemic occurred, involving 69 cases in a quite sharply circumscribed locality. In 1909 but 1 case occurred in this whole region. So that one can prophesy, to a certain extent at least, that a limited locality, severely affected during one year, will probably enjoy partial immunity in the succeeding year. On the

other hand, many localities having the disease to a moderate extent seem to go on from year to year at about the same rate.

Relative Prevalence in Smaller Cities and Towns. — Infantile paralysis is a disease affecting by preference the smaller cities and towns. This statement rests on the observation of 2,138 cases in Massachusetts which have been analyzed in this regard.

The incidence per 1,000 of the population was recorded for every affected city and town in the State for four years. For each year the cities and towns were then arranged in the order of greatest incidence, and then the first 25 on the list were contrasted with the last 25, for each year. The average population for the first 25 was then reckoned for the four years, and proved to be 5,205. The average population of the last 25 on the list, those where the incidence was least, was 52,674. A further study of the table shows that some such proportion as this existed in every one of the four years. That is to say, cities and towns where the disease was relatively least frequent were ten times as large, on the average, as those where it was most frequent. Furthermore, as a control, we tabulated the cases of scarlet fever reported in the State for the year 1910 in the same way. In the 25 cities and towns in which scarlet fever was most prevalent the average population was 6,446, and in the 25 where it was least prevalent per 1,000 of the population the average population was 7,633, practically the same, showing that scarlet fever showed no such predilection for the smaller cities and towns.

TABLE 4. — *Comparative Prevalence of Infantile Paralysis and Scarlet Fever with Reference to Population of Cities.*

DISEASE.	Twenty-five Cities where Disease was *Most* Prevalent.	Twenty-five Cities where Disease was *Least* Prevalent.
Acute epidemic poliomyelitis, ...	5,205	52,674
Scarlet fever, ...	6,446	7,633

In seeking to analyze some of the conditions which were constant in the smaller cities and towns, and which did not prevail in the larger, the number of domestic animals in each city and town was looked into, and the analysis contributes the information that the number of such animals is relatively very much greater in the smaller cities and towns than in the larger. Such a fact may have no significance whatever or it may in the future prove of importance. The table is as follows: —

TABLE 5. — *Prevalence of Infantile Paralysis with Reference to the Number of Animals in Cities and Towns.*

Total Number of Animals assessed in Twenty-five Cities and Towns where the Disease was Least Prevalent, i.e., in the Larger Cities and Towns.

Total population,	943,614
Total number of cows,	11,160
1 cow to 84.5 inhabitants.	
Total number of horses,	29,519
1 horse to 32 inhabitants.	
Total number of swine,	3,288
1 swine to 287 inhabitants.	
Total number of fowls,	41,887
1 fowl to 22.5 inhabitants.	
Total number of dogs,	23,854
1 dog to 39.5 inhabitants.	

Total Number of Animals assessed in the Twenty-five Cities and Towns where the Disease was Most Prevalent, i.e., in the Smaller Cities and Towns.

Total population,	142,639
Total number of cows,	12,801
1 cow to 11 inhabitants.	
Total number of horses,	10,352
1 horse to 14 inhabitants.	
Total number of swine,	2,374
1 swine to 60.5 inhabitants.	
Total number of fowls,	79,797
1 fowl to 1.75 inhabitants.	
Total number of dogs,	5,865
1 dog to 24.3 inhabitants.	

Of course this is only one of many conditions which are different in the two classes, but the persistence, year after year, of this greater prevalence of the disease in the smaller cities and towns must mean something with regard to the etiology of the disease if we were only able to interpret it.

Seasonal Occurrence. — The occurrence of the disease by months shows that the maximum incidence of infantile paralysis occurs between June and November. In 1907 the largest number of cases occurred in September; in 1908 July and August had equal numbers; 1909 had the largest number in August, and 1910 in July. There seems thus, to be in Massachusetts a distinct tendency of the maximum incidence to recede in point of time, a phenomenon which, should it occur in 1911, might be worthy of serious investigation.

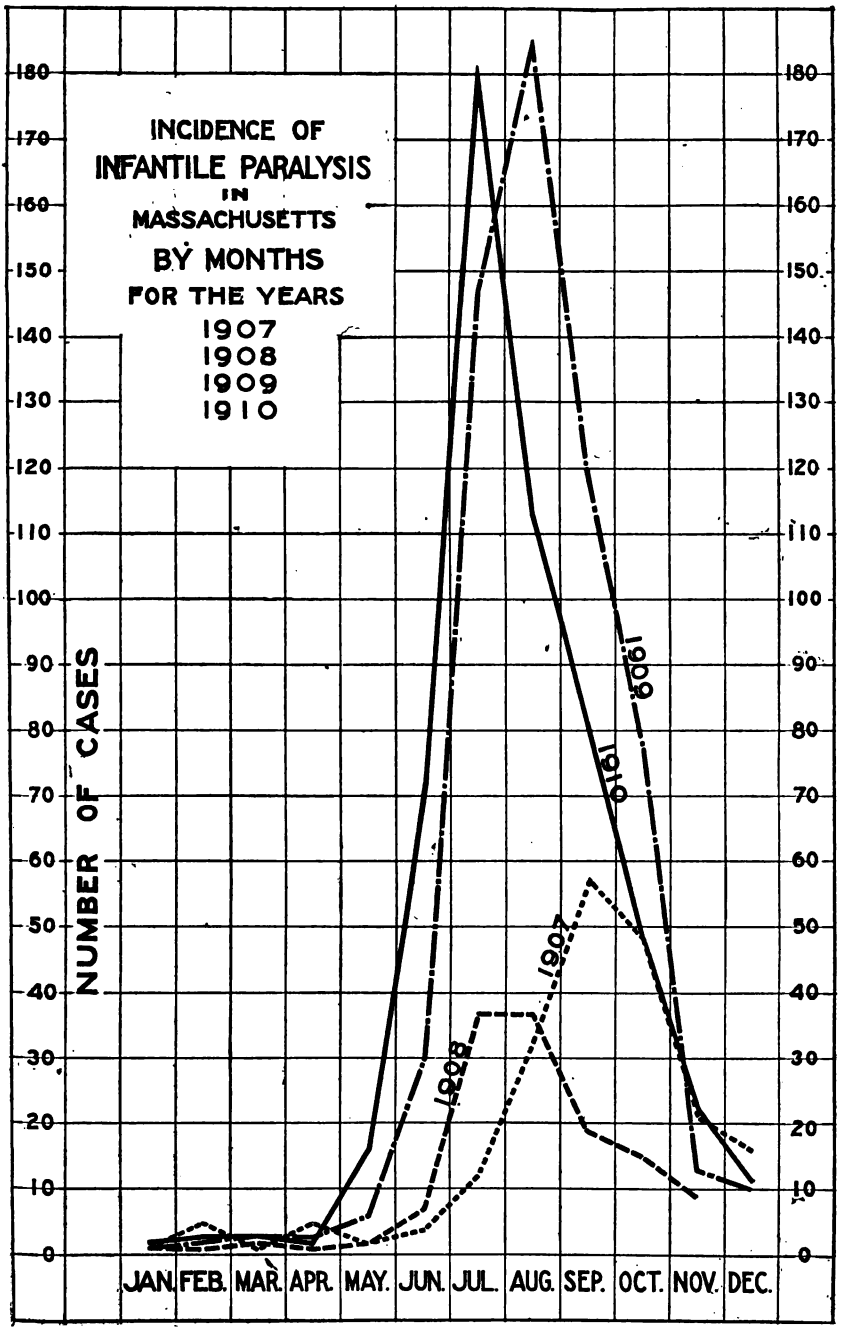

Chart 5.

Age. — The combined experience of three years, 1908 to 1910, shows that 6.13 per cent. of the cases occurred before the end of the first year, 66.94 per cent. before the end of the fifth year, and 83.1 per cent. before the end of the tenth year. Moreover, the Massachusetts figures give the impression that the disease is more frequent in adults than has been commonly supposed.

TABLE 6. — *Occurrence by Age Periods (1908–10).*

AGE (YEARS).	Cases.	Per Cent.
Birth to 12 months inclusive,	55	6.13
1,	119	13.26
2,	155	17.35
3,	125	13.93
4,	92	10.25
5,	54	6.02
	600	65.94
6 to 10, inclusive,	145	16.16
	745	83.10
11 to 20, inclusive,	83	9.25
	828	92.30
21 to 30, inclusive,	39	4.34
31 to 72, inclusive,	17	1.89
Not stated,	13	–
Total,	897	–

Sex. — As far as sex is concerned, the relative incidence is expressed by the figures of 56 per cent. for males and 44 per cent. for females, as reported for the four years 1907 to 1910.

TABLE 7. — *Sex (1907–10).*

	Cases.	Per Cent.
Males,	906	56.66
Females,	689	43.08
Not stated,	4	.25
Total,	1,599	–

Mortality. — The mortality under one year is high, being for 1909–10, 12.19 per cent. Between one year and ten years the fatality seems to drop, being 6.24 per cent., but after ten years the mortality again rises, and reaches 14.81 per cent. The average mortality is 7.9 per cent.

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TABLE 8. — *Mortality by Age (1909–10).*

AGE (YEARS).	Cases.	Deaths.	Per Cent.
Under 1,	82	10	12.19
1 to 10,	945	59	6.24
Over 10,	189	28	14.81
Totals,	1,216	97	-
Average mortality,	-	-	7.90

Environment of Patients. — As regards the character of the house, practically an equal number of cases occurred in detached houses, in tenements or in apartments. Furthermore, there seemed to be no marked predilection of the disease for any special floor of the house. The house was said to be dry in 72 per cent., damp in 27 per cent. and medium in 1 per cent. No apparent relation to moisture can, therefore, be made out. The sanitary conditions were either excellent, good or fair in 85.6 per cent. cases, and bad in only 11.5 per cent. The age of the house was not remarkable, 41.14 per cent. cases living in new houses, that is to say, within the first and tenth year of its existence, whereas 58.85 per cent. lived in houses more than ten years old.

TABLE 9. — *Character of House (1907–09).*

	Cases.	Per Cent.
Detached,	220	49.54
Tenements,	224	50.45
Total,	444	-

TABLE 10. — *Floor of House inhabited by Family (1907 and 1909).*

	Cases.	Per Cent.
The whole house,	75	21.06
The first floor,	139	39.04
First floor and basement,	3	.84
Second floor,	101	28.37
Third floor,	32	8.98
Upper stories,	6	1.68
Total,	356	-

TABLE 11. — *Location of House (1907–10).*

	Cases.	Per Cent.
Dry,	459	71.38
Damp,	177	27.52
Medium,	7	1.08
Total,	643	–

TABLE 12. — *Sanitary Condition (1907–10).*

	Cases.	Per Cent.
Excellent,	123	18.83
Good,	271	41.50
Fair,	165	25.25
Bad,	74	11.33
Not stated,	20	3.06
Total,	653	–

TABLE 13. — *Age of House (1909–10).*

	Cases.	Per Cent.
New house (one to ten years),	144	41.14
Old house (over ten years),	206	58.85
Total,	350	–

It has seemed at times as if the disease preferred river beds, but this factor in the occurrence of the disease must rest on other basis than mere propinquity, because, although 33 cases are said to have lived near a river or near water, 317 lived more than ⅛ of a mile from any stream, pond or beach, so that it seems probable that any marked occurrence of the disease along the water courses is due to other factors than a neighborhood of water, the determining influence being in all probability increased personal contact, due to the fact that all highroads, electric railways and railroads are very apt to follow river valleys, and cities and towns are, therefore, more likely also to be situated along such water courses.

TABLE 14. — *Nearness of House to Water (Stream, Pond or Beach) (1909–10).*

	Cases.	Per Cent.
On river,	33	9.42
Within ⅛ mile,	66	18.85
Within ¼ mile,	89	25.42
Within ½ mile,	78	22.28
Within 1 mile,	84	24.00
Total,	350	-

In the same way no especial relation could be found between the occurrence of infantile paralysis and nearness to railroads. The experience of Wickman, as shown by his maps, certainly seemed to show a distinct preference of the disease for localities near railroads, but in Massachusetts we have not been able to observe any etiological connection between the two. In 1909–10, 30.56 per cent. of the cases occurred within ⅛ of a mile of the tracks, while 69.42 per cent. were more than ¼ of a mile away.

TABLE 15. — *Nearness of House to Railroad (1909–10).*

	Cases.	Per Cent.
On railroad,	57	16.28
Within ⅛ mile,	50	14.28
Within ¼ mile,	44	12.57
Within ½ mile,	114	32.57
Within 1 mile,	85	24.28
Total,	350	-

A study of sewage condition shows that the disease occurs under all conditions of sewage disposal and as regards water supplies from metropolitan and city systems, as well as from wells and springs.

TABLE 16. — *Character of Sewage Disposal (1908–10).*

	Cases.	Per Cent.
Sewage (metropolitan),	110	26.25
Sewage (city, etc.),	211	50.35
Cesspool,	23	5.48
Vault,	60	14.31
Privy,	15	3.57
Total,	419	-

TABLE 17. — *Character of Water Supply (1908–10).*

	Cases.	Per Cent.
Metropolitan,	120	27.98
Town,	62	14.45
City,	172	40.09
Well,	17	3.96
Spring,	58	13.51
Total,	429	–

Rainfall. — The deficiency in rainfall during the last eight years has been, of course, very marked, and this factor may be of importance in the spread of the disease. It certainly has to be taken very seriously into consideration if we accept the suggestion of Hill of Minnesota that dust, especially dust infected with horse manure, may be responsible for the transference of this disease. The deficiency of rainfall in Massachusetts has varied since 1904, when it was 1.35 inches, to 9.42 inches in 1910. With the deficiency of rainfall during each year of this period has occurred a sort of cumulative effect, which makes this deficiency relatively more important each year. A study of the deficiency rainfall shows that the largest number of cases have not occurred on the driest years in Massachusetts.

TABLE 18. — *Deficiency Rainfall (1904–10).*

YEAR.	Cases in State.	Actual (Inches).	Normal (Inches).	Deficiency (Inches).
1904,	–	43.81	45.16	—1.35
1905,	–	37.60	–	—7.56
1906,	–	43.21	–	—1.95
1907,	234	44.49	–	—0.67
1908,	136	37.61	–	—7.55
1909,	923	42.10	–	—3.06
1910,	845	35.50	–	—9.42
	–	–	–	—31.56

Dust. — A study of the amount of dust in affected localities shows a report of a moderate amount in more than half of the cases, while in 36 per cent. of the total number much dust or an excessive amount was reported. The matter is not, however, one capable of measurement or exact determination, and the table cannot be regarded as important.

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TABLE 19. — *Relation to Dust (1909–10).*

	Cases.	Per Cent.
No dust,	–	–
Very little dust,	29	8.28
Moderate amount of dust,	195	55.71
Much dust,	118	33.71
Excessive amount of dust,	8	2.28
Total,	350	–

Insects, etc. — The possible transfer of the disease by vermin, insects, rodents, etc., was investigated, but the figures are not at all conclusive, and very little knowledge of value is likely to come in this department of the work until individual infected localities are investigated by persons expert in the study of entomology, a line of investigation of the greatest importance.

TABLE 20. — *Presence of Vermin, Insects, Rodents, etc. (1909–10).*[1]

	Families.	Per Cent.
Flies,	298	90.850
Mosquitoes,	142	43.290
Mice (house),	123	37.500
Ants,	97	29.570
Rats,	77	23.470
Bedbugs,	70	21.340
Roaches,	64	19.510
Spiders,	54	16.460
Mice (field),	24	7.310
Biting flies,	10	3.040
Fleas,	4	1.210
Grubs and caterpillars,	3	.910
Bees,	2	.609
Sand fleas,	1	.304
Snakes,	1	.304
Crickets,	1	.304
Squirrels,	1	.304
Leeches,	1	.304
Brown-tail moths,	1	.304
Moles,	1	.304
Total,	328	–

[1] Report 1907 and 1908 only for flies and mosquitoes.

Domestic Animals. — The role played by domestic animals in the transfer of the disease is one of great possible interest, but as yet the interrelation of the paralyses of man with those of animals has not been sufficiently marked to justify any definite conclusion. Many cases of paralysis in animals have been reported, but the instances in which paralysis in human beings has been coincident with such animal paralysis have been rare.

TABLE 21. — *Data as to Domestic Animals (1909–10).*

	Families.	Per Cent.
No animals of any kind in,	131	39.93
Animals in,	197	60.06
125 animals had sickness, paralysis or death in,	70	21.34
Total,	328	-

Transmissibility. — The degree of contagiousness of infantile paralysis is a question of great theoretical and practical importance. But it is one which at the present time it is practically impossible to analyze exactly because, associated with the paralytic cases of this disease, occur an unknown number of so-called abortive cases, which may be of the greatest importance in transferring to others paralytic types of the affection. Furthermore, these unknown abortive cases become immune to subsequent infection, and, if included in any study of comparative contagiousness, will go far undoubtedly to vitiate any conclusions drawn. Our figures show that in 350 cases carefully studied there was direct contact with an acute case in 13 per cent.; direct contact with possible abortive cases in 1.42 per cent.; with chronic cases in 4.28 per cent.; and that there was, undoubtedly, indirect contact with an acute case through a third person in 4 per cent. of the cases. (See chart 14 for an endemic focus in a city of 119,000 inhabitants; cases occurring in the years 1903, 1908, 1909 and 1910.)

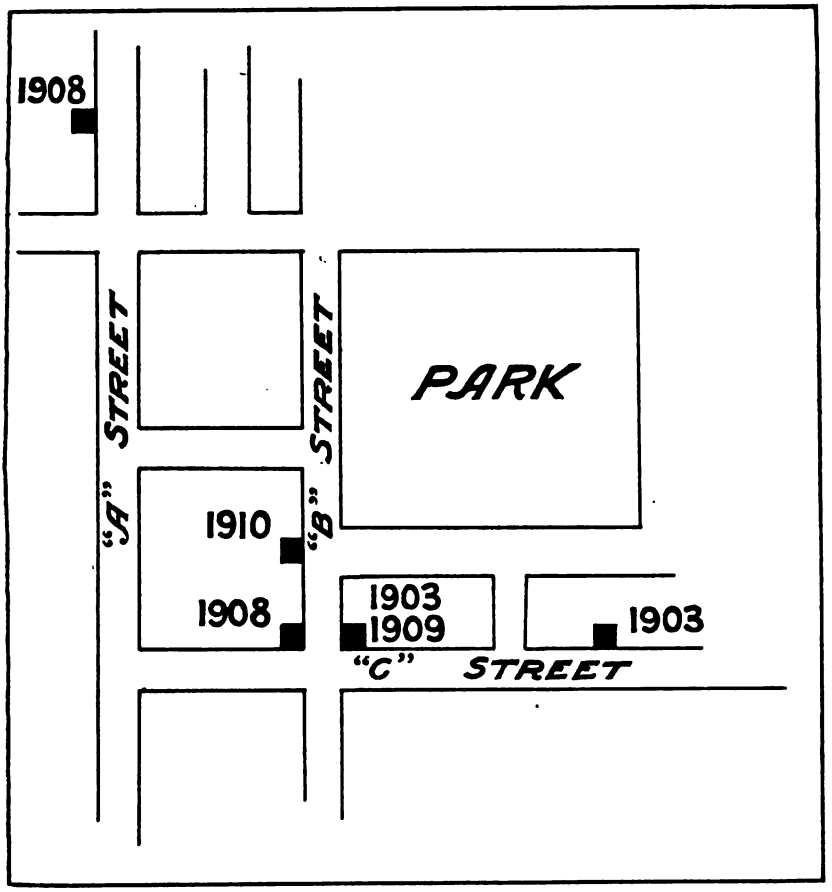

CHART 14.

TABLE 22. — *Instances of Contagiousness (1909–10).*

	Cases.	Per Cent.
Certain direct contact: —		
With acute case,	46	13.14
With possible abortive case,	5	1.42
With chronic case,	15	4.28
With both acute and chronic cases,	2	.57
Certain indirect contact with acute case by third person,	14	4.00
Number of cases investigated,	350	–

TABLE 23. — *Families with More than One Case (Reason for Suspecting Contagion) (1908–10).*

	Cases.	Per Cent.
One case,	357	92.24
Two cases,	27	6.97
Three cases,	3	.77
Total,	387	-

Institution Cases. — Forty-four institutions for children in Massachusetts were written to in 1910 in order to ascertain whether or not any cases of acute epidemic poliomyelitis had originated within those institutions during the year. One institution in Fall River, containing 360 children, reported a single case, a boy, four years old. No secondary cases occurred in this institution.

TABLE 24. — *Institution Cases.*

	Cases.	Incidence per 1,000 (Per Cent.).
44 institutions, with 3,600 children under fifteen years of age,	1	.277
Massachusetts, with a population of 851,092 children under fifteen years (census of 1905), showed in 1910 cases of acute epidemic poliomyelitis under fifteen years,	532	..625

Children in institutions, therefore, would seem to be, in some measure and in some way as yet unexplained, protected from this disease. Their opportunities for contact with the outside world are obviously much curtailed in comparison with those of the ordinary child, and therein lies very probably the reason for their immunity.

Swimming, etc. — Just prior to the disease there was a history of swimming, wading or paddling, in water more or less contaminated, in 25 per cent. of the cases just before attack.

TABLE 25. — *Swimming, Wading or Paddling (1908–10).*[1]

	Cases.	Per Cent.
Swimming, wading or paddling,	105	25.05
No such history,	314	74.94
Total,	419	-

[1] Water contaminated more or less in every instance.

Accidents. — In the same way, 34 per cent. of 457 cases gave a history of accidents, over-exertion or falls. With regard to the swimming, etc., the percentage would seem to be no higher than one would expect in any group of children at this season living near the water. But the percentage of 34 of accidents, falls and over-exertion just before the attack would seem perhaps unduly high.

TABLE 26. — *Accident, Over-exertion or Fall preceding Attack (1907–10).*

	Cases.	Per Cent.
Accident,	41	8.97
Over-exertion,	47	10.28
Fall,	67	14.66
No such history,	302	66.08
Total,	457	-

Other Diseases. — A study of diseases prevalent in affected towns coincident with infantile paralysis showed nothing of importance. A further study of recent antecedent illnesses in the patient showed merely a number of minor ailments of no especial significance.

TABLE 27. — *Diseases prevalent in Town at Time of Occurrence of Infantile Paralysis (1909–10).*

	Cases.	Per Cent.
Not known,	159	46.490
Infantile paralysis,	120	35.080
Whooping cough,	12	3.508
Measles,	11	3.210
Gastro-enteritis,	8	2.330
Diarrhœa,	6	1.750
Influenza,	6	1.750
Tonsilitis,	4	1.160
"Rheumatic fever",	4	1.160
Scarlet fever,	3	.870
Mumps,	2	.580
Malaria,	2	.580
Pneumonia,	1	.290
Coryza,	1	.290
Chicken-pox,	1	.290
Typhoid,	1	.290
Diphtheria,	1	.290
Total,	342	-

Diet. — The relation of diet to infantile paralysis very possibly is of importance but as yet no definite relation between the disease and certain kinds of food has ever been made out. Although evidently unusual the disease does occur in breast-fed babies, 4 cases of the kind having been recorded in our returns of the last two years.

TABLE 28. — *Diet (1909–10).*

	Cases.	Per Cent.
General,	283	80.85
(*a*) Raw cow's milk,	308	88.00
(*b*) Condensed milk,	15	4.28
(*c*) Breast milk and other food,	19	5.42
(*d*) Breast milk alone,	4	1.14
Fish,	265	75.71
Fruit,	288	82.28
Berries,	197	56.28
Meat,	275	78.57
Canned goods, (*a*) fruits; (*b*) vegetables; (*c*) fish,	202	57.71
Cereals,	140	40.00
Bread and butter,	23	6.57
Vegetables,	275	78.57
Stews and soups,	11	3.14
Eggs,	186	53.14
Tea, coffee, cocoa,	6	1.71
Malted milk,	2	.57
Ice cream,	83	23.71
Predigested and beef juices,	4	1.14
Candy,	27	7.71
Bananas,	6	1.71
Total,	350	–

Symptoms. — Digestive disturbances are common before, during and after the attack. Diarrhœa is less frequent comparatively than constipation.

TABLE 29. — *Details of Digestive Disturbance (1909–10).*

	Cases.	Per Cent.
Preceding attack: —		
Nausea and vomiting,	120	34.28
Constipation,	109	31.14
Colic,	5	1.42
Diarrhœa,	39	11.14
Indigestion or stomach upset,	4	1.14
Mucus in stools,	2	.57
Accompanying attack: —		
Nausea and vomiting,	140	40.00
Constipation,	149	42.57
Colic,	15	4.28
Diarrhœa,	56	16.00
Indigestion (indefinite),	3	.85
Mucus in stools,	1	.28
Following attack: —		
Nausea and vomiting;	5	1.42
Constipation,	122	34.85
Colic,	2	.57
Diarrhœa,	18	5.14
Mucus in stools,	2	.57
Total,	350	-

The analysis of the general symptoms of the acute attack in 720 cases show fever present in 88 per cent., brain symptoms in 37 per cent. and retraction of the head in 34 per cent. Sore throat was recorded as present in only 9 per cent.

TABLE 30. — *General Features of Acute Attack (1907–10).*

	Cases.	Per Cent.
Fever,	636	88.33
Pain and tenderness,	528	73.33
Brain symptoms,	266	36.94
Retraction,	248	34.44
Sore throat,	65	9.02
Total,	720	-

Appearance of Paralysis. — The appearance of paralysis in relation to the attack was studied in 589 cases. In 16 per cent. paralysis occurred on the day of the attack, in 16 per cent. one day after the attack, in 17 per cent. two days after the attack, and in 16 per cent. three days after the attack; that is to say, in 65 per cent. within three days after the attack. In 2 cases it was recorded as coming before the attack and in 23 cases (4 per cent.) between one and two weeks after the onset.

TABLE 31. — *Appearance of Paralysis in Days and Weeks after Onset of Fever (1907–10).*

	Cases.	Per Cent.
Preceding attack,	2	.330
Same day,	95	16.120
One day,	93	15.780
Two days,	103	17.490
Three days,	98	16.630
Four days,	58	9.840
Five days,	22	3.730
Six days,	51	8.650
Seven days,	18	3.050
Eight days,	6	1.010
Nine days,	2	.330
Ten days,	4	.670
Eleven days,	3	.509
Twelve days,	5	.840
Thirteen days,	1	.169
Fourteen days,	4	.670
Not known (fatal),	1	.169
Two to three weeks,	6	1.010
Three to four weeks,	1	.169
Four to five weeks,	1	.169
Eight weeks,	1	.169
Two days previous,	1	.169
Not stated,	13	2.207
Total,	589	–

Pain and Tenderness. — The great importance of pain and tenderness as a symptom is not sufficiently recognized. There were 1198 cases analyzed in this regard, and in 70.5 per cent. these symptoms were present. The disappearance of pain and tenderness in an analysis of 604

cases where the time was recorded occurred in 30 per cent. within one week, in 15 per cent. in from one to two weeks, and in 13 per cent. in from two to four weeks. In 5 cases it was recorded as lasting from two to three months, and in 3 as lasting several months.

TABLE 32. — *Pain and Tenderness (1907–10).*

	Cases.	Per Cent.
No pain,	178	14.85
Pain,	845	70.53
Not stated,	175	14.60
Total,	1,198	-

TABLE 33. — *Disappearance of Pain and Tenderness (1909–10).*

	Cases.	Per Cent.
One day or less,	11	1.820
Two days,	22	3.640
Three days,	29	4.801
Four days,	15	2.480
Five days,	14	2.310
Six days,	3	.496
A few days,	28	4.630
One week,	59	9.760
One to two weeks,	91	15.060
Two to three weeks,	46	7.610
Three to four weeks,	33	5.460
Four to five weeks,	1	.160
Six to seven weeks,	1	.160
One to two months,	28	4.630
Two to three months,	5	.820
Several months,	3	.496
Until death,	39	6.450
Present when report was made,	175	29.130
Total,	604	-

Distribution of Paralysis. — The distribution of paralysis was studied in 1,158 cases. The most common location was in one leg, occurring in 28 per cent.; both legs came next in frequency, with 24 per cent.; one or both arms in 9 per cent.; and where one arm and leg were affected a hemiplegic distribution was more common than a crossed paralysis,

the relative percentages being 10 per cent. and 3 per cent. Especial attention should be called to the frequent occurrence of paralysis of the back, abdomen, neck and face, which are much more frequent than supposed and are too often overlooked.

TABLE 34. — *Distribution of Paralysis (1907–10).*

	Cases.	Per Cent.
One leg only,	324	27.970
Both legs only,	272	23.480
Back,	154	13.290
Both arms and both legs,	129	11.130
One arm and leg, same side,	110	9.490
One arm only,	84	7.250
Both legs and one arm,	75	6.470
Face,	74	6.380
Abdomen,	67	5.780
One arm and leg, opposite sides,	33	2.840
Respiration,	31	2.670
Both arms only,	23	1.980
Neck,	11	.940
Both arms and one leg,	10	.860
Deglutition,	7	.604
Neck and back,	6	.510
Ataxia (transitory),	5	.430
General,	3	.250
Intercostal,	1	.086
Both arms, back, chest and throat,	1	.086
Total,	1,158	–

Prognosis. — The prognosis in infantile paralysis is much better than used to be supposed.

In order to throw additional light on the question of prognosis Dr. B. E. Wood of Boston was requested by the Board to investigate the present condition of all cases reported to the Board as paralyzed in the year 1907. This inquiry was made with especial reference as to the number of recoveries occurring in the four years which had elapsed. Letters were sent to all physicians who had reported cases in 1907, asking whether or not such cases had recovered, and when recovery was reported such cases were seen, unless they had disappeared or moved, and if such was the case all available information was obtained about the patients.

Of 234 cases 22 could not be investigated; 11 had died in the acute stage of the disease; 8 others had died subsequently of other diseases, although 2 of them are said to have recovered from the paralysis; no trace could be found of 3 cases. In 212 cases, therefore, the termination is known, and 57 of these (27 per cent.) are reported as having recovered.

On investigation it was found that there were three classes of recovery: (1) complete recovery without atrophy; (2) recovery with complete function but with some atrophy; (3) recovery with some hypertrophy of the affected limb. The table shows the proportion of each of these: —

TABLE 35. — *Condition of Patients after Recovery.*

	Cases.	Per Cent.
Complete recovery without atrophy,	16	28.1
Functional recovery with atrophy,	21	36.8
Recovery with some hypertrophy,	3	5.3
Recovery, presence or absence of atrophy unknown,	17	29.8

When atrophy was present the maximum amount noted was as follows: —

TABLE 36. — *Remaining Atrophy after Recovery.*

	Inches.
Calf,	$1\frac{3}{4}$
Thigh,	$1\frac{1}{4}$
Arm,	$\frac{1}{2}$
Forearm,	$\frac{3}{4}$

In most cases the atrophy was much less than this, a difference of $\frac{1}{8}$ inch being counted as atrophy, but in all such cases function of every muscle was perfect. The amounts of hypertrophy recorded were in two instances $\frac{1}{4}$ inch and in one $\frac{3}{8}$ inch, both of the calf.

In these cases the severity of the attack was classed as: —

TABLE 37. — *Severity of the Attack.*

	Cases.	Per Cent.
Severe,	14	24.5
Moderate,	9	15.9
Mild,	28	49.1
Not noted,	6	-
	57	-

The distribution of the paralysis in the 57 recovered cases was follows: —

TABLE 38. — *Distribution of the Paralysis.*

One lower extremity,	12
Both lower extremities,	9
One upper extremity,	6
Both upper extremities,	2
One lower and one upper extremity,	10
Three extremities,	7
Four extremities,	5
Face,	1
General,	1
Not given,	4

In addition to this distribution, paralysis of the face also existed in 4, of the back in 5, of the face and back in 1, and of the abdominal muscles in 1.

The time of recovery in the 57 cases was as follows: —

TABLE 39. — *Time of Recovery.*

1 week or less,	2
1 week to one month,	8
1 to 2 months,	8
2 to 3 months,	5
3 to 6 months,	10
6 to 12 months,	9
1 to 2 years,	5
2 to 3 years,	5
No data	5

CONCLUSIONS AS TO PROGNOSIS.

The following conclusions seem justified as regards prognosis: —

Complete recovery or functional recovery occurs in something over 25 per cent. of cases examined at the end of four years. Atrophy may exist without impairment of function. In about half of the recovered cases the onset was mild. The distribution of the paralysis in such cases was not essentially different from that of cases which do not recover. The period of recovery in many instances occupied months and in several cases from one to two years.

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A STUDY OF AN EPIDEMIC OF INFANTILE PARALYSIS (ACUTE EPIDEMIC POLIOMYELITIS) IN SPRINGFIELD, MASS., IN 1910.[1]

By Philip A. E. Sheppard, M.D., Boston.

An account of all the cases of infantile paralysis reported in the State, including cases in and about Springfield, is to be found in a report to the Massachusetts Board of Health;[2] in the present study, however, are comprised 200 cases.

A description of the city of Springfield is, in brief, as follows: —

Population by United States Census, 1910, 88,926, composed mainly of the more prosperous classes of mechanics, store and office people, manufacturers and merchants. It is estimated that nearly 3 per cent. of the population are commercial travelers.

A large percentage of the inhabitants resides in detached one and two-family houses, the flat dwellers being in a very small minority. With the exception of those in Indian Orchard, there are no "mill" tenements. There are many churches in Springfield. Many of the fraternal societies have a lodge or council. The city has numerous men's, women's, boys' and girls' clubs. Many social, whist and kindred clubs meet daily at the homes of the members. Many of the fraternal orders maintain social rooms.

Four theatres and ten moving-picture houses are crowded at nearly every performance (all but two were open during the summer 1910). The various dances, athletic exhibitions, etc., are well patronized.

The city has several large playgrounds. In addition, many school houses and grounds are used for purposes of entertainment during the summer months, while thousands visit Forest Park (the chief public park) daily.

The transient population is extensive. It is estimated that Springfield is the trading center for 500,000 to 600,000 people. A large number of automobile tourists are entertained at the various hotels each day. Hundreds of the working people reside in nearby cities and towns, and many Springfield people are employed in other places. The railroad and trolley lines entering the city are patronized probably far in excess of those in other cities of equal population. (No unusual increase in freight or passenger travel during 1910.)

[1] Report submitted to State Board of Health, May 1, 1911.

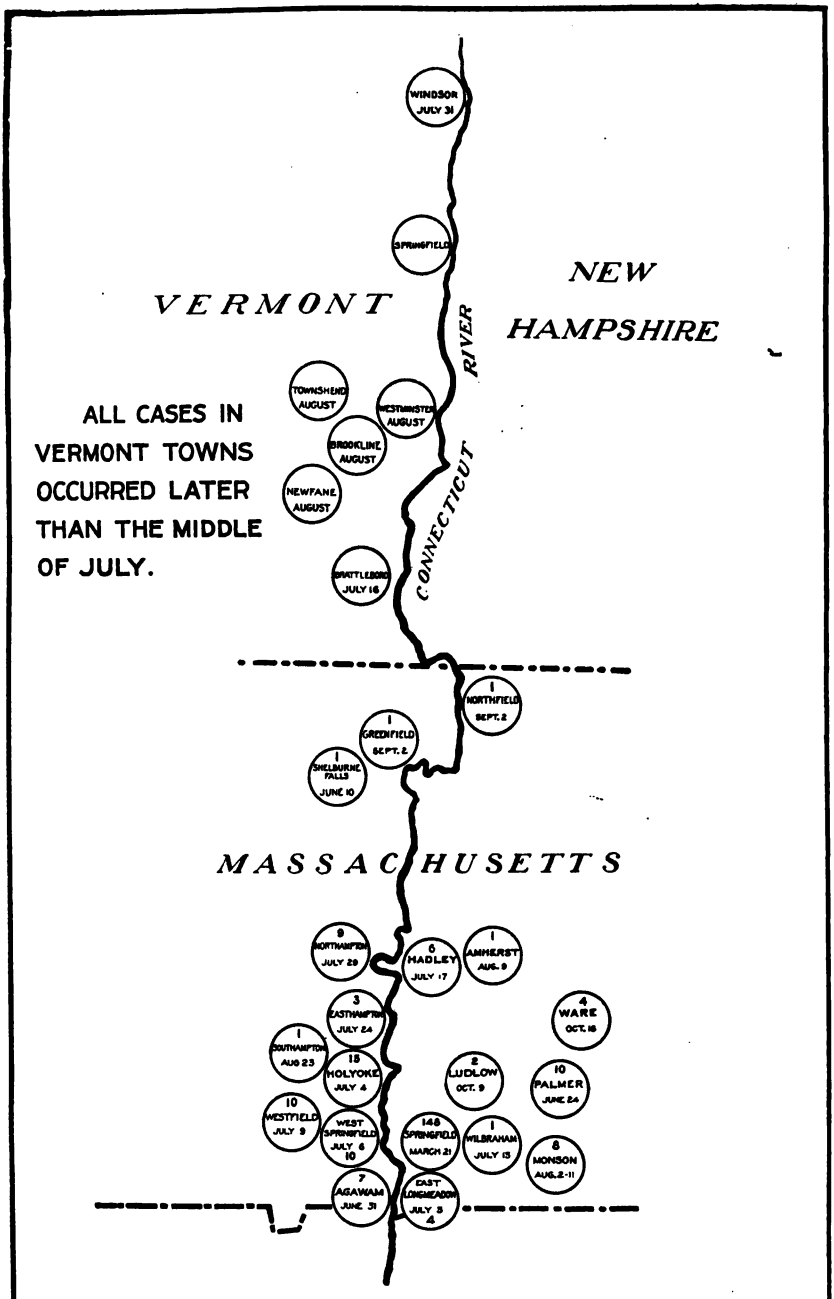
[2] Lovett and Sheppard: Occurrence of Infantile Paralysis in Massachusetts, Boston Medical and Surgical Journal, May 25, 1911.

The industries of the city are varied. Firearms, railroad cars, automobiles, machine tools, machinery, papeteries, skates, soap, wire, buttons and art goods are the more important articles of manufacture. Several large publishing houses and a large number of stores and offices furnish employment to thousands.

General Conditions affecting Springfield and the Neighboring Towns.

The Connecticut River Valley. — It has been thought by some observers that the incidence of the disease was greatest in the cities along the river beds. From the Massachusetts maps for 1907, 1908, 1909 and 1910, it would certainly seem that this idea is not without foundation. That there is some association of the disease with the watercourses seems probable. A study of the maps of Massachusetts for the four years above named will show at once a grouping of cases along certain of the river beds, such as the Connecticut, the Deerfield, the Merrimac, the Housatonic and the Hoosick rivers, although in addition small scattered groups of cases have always been found throughout the State. In 1910, however, the chief epidemic focus in the State of Massachusetts was located in Springfield, and it may serve a useful purpose to give a short description of the Connecticut River bed at this point, though the reasons for the incidence of the disease in this city are difficult to determine. The Connecticut River has its source in the Connecticut lakes in northern New Hampshire. Its extreme headwaters, however, lie in the Province of Quebec, in which province an epidemic of poliomyelitis is said to have occurred previous to that in Springfield and in Vermont, and in the mountains of the northern boundary of New Hampshire and Vermont, and its course lies through Massachusetts and Connecticut to Long Island Sound. It is the largest river in New England, except the St. John, has a total length of about 345 miles and a total drainage area of 11,085 square miles, of which about 155 square miles lie in the Province of Quebec. The Connecticut has numerous important tributaries. From its headwaters to the Sound the Connecticut falls about 1,900 feet. In Massachusetts the Deerfield and Westfield rivers with their sources in the Berkshires are quick-spilling streams, with steep slopes and narrow valleys largely wooded, while on the east the Miller's and Chicopee rivers drain a flatter country with numerous ponds and reservoirs. The rocks in the Connecticut valley are, in general, red sandstone, extending from New Haven nearly to the northern boundary of Massachusetts. The prevailing surface material is glacial drift. The mean annual precipitate in the Connecticut valley is about 40 inches.[1]

[1] Progress Report on the Hydrographic Investigations in Massachusetts, 1909 to 1910, by C. C. Covert.



SPREAD OF INFANTILE PARALYSIS ALONG CONNECTICUT RIVER VALLEY.

(NUMBER OF CASES AND DATE OF FIRST CASE SHOWN FOR EACH AFFECTED CITY OR TOWN.)

No exact data as to the distribution of the disease in the States of New Hampshire and Vermont are available, but cases have been reported in both these States. If, as is supposed, the disease travels along the watercourses, then it might possibly be shown that pollution of the stream occurred higher up the river, or that insects, such as mosquitoes or biting flies, played an important role in the etiology of the disease, or, more simply, that the greater relative density of population explained the greater incidence along the river beds. (See chart.)

The following table shows the incidence of the disease in the city of Springfield for the past few years as reported to the local board of health.

Year.	Cases.	Incidence per 1,000.	Year.	Cases.	Incidence per 1,000.
1907, ...	2	.024	1909, ...	1	.012
1908, ...	3	.035	1910, ...	148	1.670

For this last year (1910) my estimate of the number of cases reported, missed and abortive, is 300. (I have used the word "reported" for each of these years advisedly, because if the history of chronic cases is thoroughly gone over for this city for the four years, 1907–10, I am convinced that the incidence of cases will be shown to be larger.)

Deaths from Various Causes in Springfield during 1910 by Months.

Disease.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Totals.
Cerebro-spinal meningitis, .	–	–	–	–	1	1	1	–	–	–	–	–	3
Diphtheria, ...	10	2	1	3	4	3	–	2	2	3	5	3	38
Tetanus, ...	1	–	1	–	–	–	–	–	1	1	–	–	4
Cholera infantum, . . .	4	4	1	3	3	17	27	20	17	6	2	2	106
Whooping cough, . . .	–	–	2	1	–	3	5	3	3	1	3	2	23
Anterior poliomyelitis, . .	–	–	–	–	–	3	14	3	3	1	1	–	25
Scarlet fever, ...	2	3	8	3	1	2	–	–	–	–	–	–	19
Measles, ...	–	–	2	–	6	1	3	–	–	–	–	–	12
Influenza, ...	4	–	4	6	2	–	–	–	–	–	–	–	16
Typhoid fever, ...	–	1	2	–	2	2	1	–	1	1	1	2	13

Deaths from Various Causes in Springfield during the Years 1907, 1908, 1909 and 1910.

Disease.	1907.	1908.	1909.	1910.
Cerebro-spinal meningitis,	4	2	1	3
Diphtheria,	20	24	33	38
Tetanus,	?	?	-	4
Cholera infantum,	25	26	28	106
Whooping cough,	13	2	3	23
Anterior poliomyelitis,	?	?	-	25
Scarlet fever,	9	8	-	19
Measles,	17	1	7	12
Influenza,	18	22	-	16
Typhoid fever,	20	22	11	13

The death-rate from a number of the other more common infectious diseases in Springfield during 1910 and the three years previous offers a number of points of especial interest. It is not surprising, of course, that cholera infantum should show an increase during the summer months similar to that of epidemic poliomyelitis, but when the total death-rates for these infectious diseases for 1910 is tabulated, the striking fact becomes plain that not only "anterior" poliomyelitis but also many other infectious and contagious diseases were very fatal in 1910. This fact becomes especially prominent when this death-rate for 1910 is compared with that of previous years (see table).

The increase in the death-rate from cholera infantum is especially striking, being practically 300 per cent. In order to explain this remarkable increase of mortality two theories suggest themselves: (1) that there was some wide-spread general influence which made the children of Springfield especially susceptible to all infectious diseases in general; or (2) that a considerable number of cases of cholera infantum, for instance, may have been gastro-intestinal types of epidemic poliomyelitis.

It will be extremely interesting if, when the figures for 1911 become available, it appears that the death-rate from those infectious diseases has dropped back to the general average maintained in the years 1907, 1908 and 1909.

A description of Springfield by wards is given hereunder with special regard to the character of the population and the industries.

Total Population, 88,926.

Wards.	Population.	Cases.	Incidence per 1,000.
Ward 1,	16,274	34	2.089
Precinct A,	7,761	–	–
Precinct B,	1,574	–	–
Precinct C,	2,965	–	–
Precinct D,	3,974	–	–
Ward 2,	10,825	16	1.478
Precinct A,	4,585	6	1.308
Precinct B,	6,240	10	1.602
Ward 3,	5,369	4	.745
Precinct A,	2,337	1	.427
Precinct B,	3,032	3	.989
Ward 4,	9,170	9	.981
Precinct A,	4,709	5	1.061
Precinct B,	4,461	4	.896
Ward 5,	7,950	11	1.383
Precinct A,	3,438	–	–
Precinct B,	4,512	–	–
Ward 6,	9,356	12	1.282
Precinct A,	4,320	6	1.388
Precinct B,	5,036	6	1.310
Ward 7,	14,258	23	1.613
Precinct A,	2,787	2	.717
Precinct B,	3,596	21	1.830
Precinct C,	4,018		
Precinct D,	3,857		
Ward 8,	15,724	21	1.330
Precinct A,	5,004	13	1.381
Precinct B,	4,401		
Precinct C,	6,319	8	1.266

Ward 1. — Thirty-four cases reported. Manufacturing and residential section. Population principally better class of mechanics, motormen and street railway conductors.

Ward 2. — Sixteen cases. Manufacturing, mercantile and residential. Heterogeneous population; Hebrews predominate. Precinct B, 10 cases. Jewish, Turkish and Syrian quarter. Rag pickers, peddlers, junk dealers, etc.

Ward 3. — Four cases. Manufacturing, mercantile and residential. Precinct A, 1 case. Population, mechanics and laborers, negro quarter, store and office people in hotels and better class of boarding houses. Precinct B, 3 cases. Population, mechanics and laborers, a portion of the Italian quarter, store and office people in better class of boarding houses and hotels. Most of the larger hotels are in this ward; large transient population.

Ward 4. — Nine cases. Mercantile and residential. Precinct A, 5 cases. Population, small portion laborers, balance merchants, manufacturers, mechanics, store and office people. Precinct B, 4 cases. Population principally better class of mechanics.

Ward 5. — Eleven cases. With exception of a few stores and Springfield Armory, wholly residential. Population, merchants, manufacturers, professional people and mechanics. Well-to-do as a rule.

Ward 6. — Twelve cases. Manufacturing, mercantile and residential. Precinct A, 6 cases. Population, principally Italian laborers. West section, Italian quarter. Precinct B, 6 cases. Merchants, manufacturers, store, office people and mechanics.

Ward 7. — Twenty-three cases. Residential wholly (except United States watershops). Precinct A, 2 cases. Principally mechanics. Precincts B, C, and D, 21 cases. "Forest Park District," merchants, manufacturers, professional, store and office people; large number commercial travelers. In comfortable circumstances as a rule. Forest Park is situated in Precinct D.

Ward 8. — Twenty-one cases. Manufacturing, farming and residential. Precincts A and B, 13 cases. Store and office people and mechanics. Precinct C, 8 cases. Large portion farming district. Population principally, in Indian Orchard, French Canadian and Polish mill operatives. Average mill-town conditions.

For Ward 5, with a population of 7,950, 11 cases were reported. In this ward is the Springfield Armory, where a fatal case occurred in the child of one of the orderlies. Investigation of this death was impossible owing to the attitude of the United States army authorities.

In Ward 7, where 23 cases occurred, the total population being 14,285, the following account of paralysis in monkeys was obtained from the superintendent of Forest Park: —

History of Paralysis in Monkeys (Macacus Rhesus) in the Forest Park Zoo in Springfield.

1904, 1 male sick with paralysis 1 month, chloroformed.
1905, 1 female sick with paralysis 2 weeks, chloroformed.
1905, 1 female sick with paralysis 2 months, recovered.
1907, 1 female sick with paralysis 22 weeks, partially recovered.
1908, 1 female, "Susie," sick with paralysis, partially recovered.
1909, 1 male sick with paralysis, partially recovered.

The sickness and paralysis in these monkeys from 1904 to 1909 all occurred in late October of their respective years. No history of tuberculosis was obtained in these animals. They were shipped to Springfield directly on arrival at New York. In "Susie's" case the affection, which resulted in a paralysis of her hind quarters, started out with convulsions, during one of which she became totally blind and remained so for two weeks. The last two monkeys, those of 1908 and 1909, were presented to me by Mr. Ladd, the superintendent of Forest Park Zoo, and given in turn by me to Drs. Osgood and Lucas, who have used them in their experimental work. Both these monkeys were inoculated with the virus of acute epidemic poliomyelitis, and both succumbed to the disease within a few days. Pathological examination showed evidences

of typical epidemic poliomyelitis in the spinal cord. Furthermore, the chronic lesions of the cord were in no sense typical of epidemic poliomyelitis in the past. The fact that these monkeys were in no way refractory to inoculation of the virus of epidemic poliomyelitis strengthens still further the conclusion that the original disease was of an entirely different nature, inasmuch as in the great majority of instances survival after inoculation and infection has produced to a greater or less extent resistance to the disease.

Table of Cases in Springfield and Indian Orchard, 130 of which were reported to the Local Board of Health in 1910, and their Time-relation to Local Festivities.

Festivities in Springfield in Brief.	Date of Onset.	Cases.
	March 21,	1
	May 5,	1
	May 8,	1
	May 20,	1
	May 22,	1
May 30, Memorial day parade.	May 25,	1
	June 3,	1
	June 4,	1
June 8, Wild West show.	June 7,	1
June 8-10, Wonderland, an out-door fair.	June 9,	1
	June 11,	2
	June 12,	3
	June 13,	1
	June 14,	2
	June 15,	2
	June 16,	1
	June 18,	2
	June 19,	3
June 20, Barnum and Bailey's circus.	June 20,	2
	June 21,	2
	June 22,	1
	June 24,	2
	June 26,	4
	June 27,	1
	June 28,	2
	June 29,	5
	June 30,	3
	July 1,	3
	July 2,	1
	July 3,	2
July 4, Celebration and parades.	July 4,	2
	July 5,	1
	July 6,	1
	July 8,	1
	July 9,	1
	July 10,	6
	July 11,	1
	July 12,	5
	July 13,	5
	July 14,	7
	July 15,	4
	July 16,	2
	July 17,	1
	July 19,	3
	July 20,	3
	July 21,	1
	July 22,	1
	July 23,	2
	July 26,	2
	July 27,	2
	July 28,	2
	July 31,	3
	Aug. 1,	4
	Aug. 3,	1
	Aug. 4,	1

Table of Cases in Springfield and Indian Orchard, etc. — Concluded.

Festivities in Springfield in Brief.	Date of Onset.	Cases.
	Aug. 6,	2
	Aug. 7,	1
	Aug. 8,	2
	Aug. 10,	2
	Aug. 11,	1
	Aug. 12,	1
	Aug. 14,	1
	Aug. 15,	1
	Aug. 16,	1
	Aug. 17,	1
	Aug. 18,	1
	Aug. 19,	2
	Aug. 21,	1
	Aug. 23,	1
	Aug. 25,	1
	Sept. 16,	1
	Sept. 20,	1
	Sept. 25,	2
	Oct. 2,	1
	Oct. 5,	1
	Oct. 15,	1
	Nov. 23,	1
	Nov. 27,	1
	Nov. 29,	1
	Dec. 4,	1
Total,		146

The festivities mentioned in the above table were in brief as follows: —

May 30. Usual memorial day parade. People out in considerable numbers.

June 8. Miller Bros. 101 Ranch Wild West. Parade in the morning, witnessed by a large crowd. Two performances; large audiences. Shows of this character do not, as a rule, draw many patrons from surrounding towns.

June 8–10. "Wonderland." An out-door fair conducted by local people for the benefit of the Boys' Club. Well patronized. Large number of children. A convention of commercial travelers was in session in Springfield June 9 to 11, and 700 or 800 attended the above-mentioned attraction on the evening of June 10. With this exception patronage was confined mainly to local people. About 900 of the commercial travelers paraded June 11. No unusual number of people on street.

June 20. Barnum and Bailey circus. Parade in morning. Streets crowded. Two performances; crowded houses at both. Probably 50 per cent. of audiences from surrounding towns. Circus arrived on morning of the 19th (Sunday). Large crowd on grounds all day.

July 4. "Safe and sane" celebration. Parades, athletic exhibitions, boat races, band concerts, fireworks, etc. All sections of city had some form of entertainment. Apparently most of the local people and thousands from out of town were on the streets or in the parks.

The five-weeks period (May 30 to July 4), therefore, offered unusual opportunities for contact, not only between human beings, but also between human beings and animals.

Springfield's New Water System. — The Little River water supply has a watershed of some 48 square miles of hills and valleys with innumerable springs and mountain brooks. Habitations are said to be scarce on the watershed, and the possibility of contamination slender. A thorough system of filtration has been installed about 16 miles from Springfield. The mains used in the late Ludlow supply were utilized, but the course of the water was reversed by the new Little River high-pressure system. The growth of algæ in these pipes was such that periodic flushings were necessary at the outset, and not infrequently the mains burst and flooded various sections of the city.

Meteorological Observations. — It is interesting to note that the conditions during July, the month of the greatest incidence of the disease in Springfield, were favorable for the production of dust. The mean range of temperature was higher for this month than for any other month but March; the mean temperature of the month was considerably higher in July than in any other month of the year, being 73.66; the mean pressure (reduced to 32° F.) was lower than in any other month; the relative humidity was less; and the total rainfall was low.

Contemporary Animal Sicknesses. — In June, 1910, my attention was directed to a small epidemic among dogs in Sixteen Acres, just outside of Springfield. The disease was characterized by a form of paralysis involving usually the hind quarters. Six dogs were thus affected, but there were no cases of acute epidemic poliomyelitis in the immediate neighborhood.

In Charlemont a horse was said to have been paralyzed. I was not able to see this animal, but the evidence came from a reliable source. There were cases of poliomyelitis in Charlemont.

In Northampton a two-year-old colt with no previous history of sickness was seized with fever and within twenty-four hours presented a paralysis involving the hind quarters. A case of acute epidemic poliomyelitis occurred within a short distance of the barn where this colt was kept. The history showed that about May 15, 1910, the colt was found one morning with paralysis of hind quarters. She had been well the day before. On June 27 the colt was enough better to make a trip to Southampton, walking the entire distance. At that time, though the colt's condition was better than before, she still had some fever, was excitable and irritable, and very sensitive to touch about the head and along the spine from withers to tail. Treatment: applications of ice and ice water to spine, diuretic, laxative and tonic (nux vomica and Fowler's solution). On September 22, colt carried her back a little humped, but gait in forelegs appeared natural. In hind quarters there was considerable ataxia. The spine was evidently sensitive to pressure along back,

but head not sensitive. There were no marks of injury. Later on the colt was sold in Connecticut as a brood mare, but was returned to its previous owner on account of impaired locomotion. Finally, I secured the colt and had it shipped to the antitoxin laboratory in Forest Hills, where a few days after its arrival it died and was autopsied by Dr. Theobald Smith. There was nothing found in the cord suggestive of the lesions of epidemic poliomyelitis.

Medical Inspection of Schools in Springfield. — This work has greatly increased lately. Twelve inspectors are employed and 41 school buildings are visited twice weekly. During the spring of every year an inspection of teeth, tonsils and adenoids is made, which involves an examination of every child attending the lower grades of public schools.

Tonsils and Adenoids. — During the spring of 1910 11,858 children were examined, and 3,538 children were found to have enlarged tonsils, and 1,635 children adenoid growths. Parents are notified by printed statement under the system employed in Springfield, in which the condition is explained and operative remedial measures are advised. Over 700 children were operated on for tonsils and adenoids at the different hospitals during 1910, and a careful inquiry among physicians showed a large number of private operations.

My interest was aroused along this line on account of one or two cases that presented a clinical picture of an acute infection involving the upper respiratory passage with some gastro-intestinal disturbances, in which paralysis seems to have been precipitated by tonsillectomy. Since the exact function of the tonsil is still a matter of speculation, I have sought to collect some data as to the possible significance of its presence or absence in cases of acute epidemic poliomyelitis.

Case 1. — Five years old. Male. Acute febrile onset July 13, 1910. On the morning of July 14 was operated on for tonsils and adenoids, which, though slightly inflamed, were comparatively normal. The following night, July 15, paralysis of both legs was noticed. On July 4 the child had an attack of vomiting after eating ice cream, which cleared up entirely before the febrile onset on July 13; during and following the febrile attack there was diarrhœa, lasting until the 19th of July. The question in this case is whether or not the inflammatory reaction of the tonsils was nature's effort to throw off the infection, and whether by depriving the child of his tonsils possibly the paralysis was precipitated.

Case 2. — Aged six, American. On Sept. 13, 1910, this child was operated on for tonsils and adenoids, and had a sore throat up to the date of his febrile onset, Sept. 25, which was accompanied by aphonia and disphagia. Paralysis of the neck muscles appeared on the 1st of

October. This child was exposed directly to another case of acute epidemic poliomyelitis before and after tonsillectomy and adenectomy were performed. The question in this case is whether or not the infection with poliomyelitis virus was acquired after the operation on the tonsils and adenoids on September 13, and it cannot well be answered. Case 2 is a typical case, and it might be reasonable to suggest at this time that tonsils and adenoid tissue (tonsillar ring of Waldeyer) may offer a resistance to microbic invasion. If this is the case, it might seem on *a priori* grounds injudicious to remove an inflamed tonsil, especially in the presence of an acute epidemic infection characterized in many instances by involvement of the upper respiratory passage.

In Springfield, before and during the epidemic of acute epidemic poliomyelitis, it is of interest to note from the above data that many operations on tonsils and adenoids were performed.

Early Cases in Springfield. — One of the earliest cases (not reported), that may have influenced the spread of the disease, occurring in Springfield in 1910, as far as I have been able to ascertain, was that of a boy of ten years, whose father was a public collector of accounts. This case had its onset late in February or early in March. The father's route was fairly general over Springfield, and necessitated repeated calls at houses where accounts were being paid off by installments. Several cases of infantile paralysis are said to have occurred in houses visited by this man subsequent to the case in his family.

Another early case was that of an infant in a family of a physician, who previously had attended the above and other cases, which had never been reported nor isolated. Later in the epidemic two other children of this same physician became affected, — a total of three cases in this one doctor's family.

Other early cases that might be mentioned are a case in another physician's family; a case in the family of a laundryman, who, on his route in the city, called at about 500 houses; two cases in the family of a Greek fruit vender, who peddled his fruit from the team and covered a considerable area of the city daily. The first case in this man's family was not diagnosed until the second had appeared, some three weeks after. On several occasions during their convalescence the fruiter's children accompanied him on his daily rounds.

From the table of cases occurring in Springfield and Indian Orchard it would appear that the epidemic began late in May, but cases occurred earlier, in fact, as early as February. These cases were neither reported to the board of health, nor were they isolated. Terminal disinfection was not carried out. In other words no precautionary measures from a public health point of view were taken with these cases, one of which

proved fatal. I visited this fatal case in order to arrange for an autopsy, and found this child laid out on the front porch and covered with flies of several varieties. Such a case could form an excellent focus for the spread of infection, provided the means for dissemination were present.

As to the incidence of the disease in Springfield as compared with that for the whole State, it would seem that no material difference existed, the greatest incidence for both occurring in July. It might be argued from this that the results of attempts at isolation and quarantine were practically nil, for if isolation had been effective in controlling the spread of the disease, then we might reasonably have looked for a decrease in the number of cases after enforcing this order. As a matter of fact, this did not occur, and may be accounted for in any one or other of the following ways.

The first isolation period declared by the Board of Health was for two weeks after the case was reported; later a four-weeks isolation period was declared. It may be that the two-weeks isolation period was not sufficiently long, nor enforced early enough to be effective. Again the four-weeks isolation period for the same reasons seems not to have been effective in checking the spread of the disease. Neither of these isolation orders applied to the cases that occurred prior to their going into effect, nor did they affect actual cases of paralysis that were never reported, occurring earlier than May, to say nothing of the number of suspicious contemporary illnesses occurring in Springfield at this time, among which may have been a considerable number of abortive cases. Furthermore, isolation measures are probably not sufficiently stringent. With absolute quarantine we might have had a very different showing. It is reasonable to suppose, then, that isolation in this instance did not effectively control the spread of the disease, since it was not enforced early, nor did it restrict the movements of the abortive cases, missed cases, and possibly "healthy" carriers.

The following circulars were sent out. A copy of the circular sent to doctors is given below: —

HEALTH DEPARTMENT, SPRINGFIELD, MASS.

ANTERIOR POLIOMYELITIS.

The State Board of health has declared anterior poliomyelitis to be a disease dangerous to the public health, and as such must be immediately reported to the local board of health. Failure to comply with this statute involves liability to a fine of $50 for each offence.

From all information obtainable of epidemics of this disease in other places, as well as a study of the epidemic now in progress in this city, the disease is without doubt of an infectious and contagious nature. The causative agent is not known, but the mortality and subsequent paralyses and deformities,

which are only too much in evidence, place anterior poliomyelitis amongst the diseases most dangerous to life and future usefulness.

The board of health has therefore established a strict isolation period for this disease of four weeks from date of notification, with disinfection at termination of the period. No public funerals will be allowed of patients dying from this disease.

It is only, however, through the earnest and active co-operation of the medical profession, who come in personal contact with the disease, that definite results can be obtained by the board of health in checking the spread of the epidemic.

Physicians are, therefore, urged to use every precaution in preventing its spread which is now in use in the management of other contagious diseases, and to explain very carefully to the family the dangers arising not only to themselves, but to others, from a disregard of the regulations of the board of health. If this line of action is pursued by the medical profession, it is believed that as good results will be obtained as to undertake the establishment of an absolute quarantine, with all its discomforts, hardships, and consequent tendency on the part of the public to conceal the existence of the disease.

If physicians will advise all their families, as a matter of self-protection, to at once establish a voluntary isolation for themselves, the best quarantine possible will then be produced. Children should be kept at home and not allowed to attend picnics, excursions, Sunday School, theatres, playgrounds or other places where children come together in numbers.

JULY 27, 1910.

HEALTH DEPARTMENT, SPRINGFIELD, MASS.

ANTERIOR POLIOMYELITIS (INFANTILE PARALYSIS).

This disease is contagious, and is followed in a great many cases by paralysis of arms, legs, or other parts of the body. Deformities with inability to use the arm or leg are liable to be permanent.

It attacks young children chiefly, but adults are by no means exempt. It is therefore of the utmost importance that you use every means possible in preventing other children from contracting this disease.

No one will be allowed to see the patient except the necessary attendants and the physician. The patient must be cared for in a room isolated from the other members of the family during the entire period of isolation. Mild cases which are only sick for a few days, and do not show any definite paralysis, are just as liable to convey the infection as the more serious cases, and must be isolated for the full period. Every case should remain under the care of your physician during the entire isolation period, which is four weeks from the date of notification sent to the board of health. Disinfection will then follow and the card be removed.

If the regulations of the board of health are not observed and the patient for any reason is not or cannot be properly isolated, the board may cause

such patient to be removed to the isolation hospital. The importance of obeying these instructions cannot be overestimated, as a large percentage of children who contract this disease will be deformed or crippled for life with consequent inability to work or provide for themselves.

It is a duty you owe to your children and those of your neighbors to exercise every precaution in preventing the continuance and spread of this disease, and unless you exert yourself in this matter, an absolute quarantine of all cases of anterior poliomyelitis will be declared by the Board of Health, which will inflict great hardship and distress not only upon yourself, but upon the entire city.

Human life is the most valuable asset we possess, and it must be protected at any cost.

It was further suggested that all children's gatherings be discountenanced by the local authorities, such as for example, picnics, excursions, public play-grounds, theatres, shows, Sunday Schools, etc.

That the clothes and linen used by the household be disinfected and excluded from the public laundry.

That the school committee be advised to extend the vacation a fortnight, thus postponing the reopening of schools until the 19th of September (the school committee were unanimous in their vote on this question, granting the extension of the vacation, and further agreed that cases of epidemic poliomyelitis, that had occurred in Springfield that summer, be excluded from the public schools for at least two or three weeks after the isolation order was raised), and that a special placard be posted on all the houses in a conspicuous place. The following is the form used in Springfield: —

KEEP OUT.

Anterior Poliomyelitis.

All persons, not inmates, are forbidden to enter this building.

BY ORDER OF THE BOARD OF HEALTH.

Any person removing this card without authority is liable to a fine of from $10 to $100.

From the beginning of May to early in July few cases of infantile paralysis occurred outside of Springfield. The disease began to appear in neighboring towns about the middle of July. Most, if not all, of the cases occurring in neighboring towns gave a history of having visited Springfield and having mingled in the crowd July 4, or at some of the festivities mentioned above.

During the early period of the epidemic many families became

alarmed and left the city for other neighborhoods. During October one of these families returned. A child seven years old, who had been apparently well while away from Springfield and was never exposed, as far as known, until it returned to the city, became ill seven days after exposure and contact with a patient in Springfield sick with infantile paralysis. Paralysis in this case, however, proved transitory, and within three months the child had recovered.

Possible Abortive or Atypical Cases, or Suspicious Contemporary Illnesses. — I have kept in mind during the epidemic in Springfield this type of the disease, and for that city in 1910 I should make a conservative estimate that at least 300 cases of all types occurred, including the abortive cases (which latter, of course, were never reported). The actual number of paralytic cases in Springfield investigated by me numbered 146. The abortive cases, as a matter of fact, are almost invariably "missed," unless they occur in a family along with a paralytic case during the course of an epidemic.

Communicability. — The following groups of cases are submitted as showing the line of contact, direct and indirect, that was traced in several instances. The case numbers agree with those shown on the map of the Springfield cases.

Group I.

Seven cases occurred in this group.

On March 21, 1910, a case occurred in the family of Mr. Y., a public collector of accounts, who reports that since the illness of his son (Case 1), aged five, several cases have developed in families where he called for the collection of accounts. Dr. X. attended this case.

Case 31 occurred in Springfield and had been in intimate contact with case 1.

Case 31, aged four, although living with his grandparents on another street, frequently visited his parents, who lived in the same house on the floor below the Y. family, where case 1 occurred. Contact was intimate and frequent between case 1 and case 31. On June 22, 1910, case 31 began his acute attack, which terminated fatally within a week. The above two cases occurred in the city of Springfield.

Dr. X. attended a case in West Springfield, not shown on the Springfield map, which had its acute onset on July 6 and terminated fatally within a few days.

On July 15 case 87 occurred in the infant daughter of Dr. X., a right hemiplegia, and at this time was not recognized as acute epidemic poliomyelitis. Dr. X., being the school physician, examined some 1,800 to 2,000 children on the reopening of schools in September. Among these he reports having seen several recovered cases, and also examined children from families where cases had occurred.

Case 143, another daughter of Dr. X., aged three, occurred on the 23d of November, 1910, left facial paralysis, and was diagnosed as Bell's palsy.

Case 145, the oldest daughter of Dr. X., aged five, occurred on Nov. 29, 1910, and was diagnosed as acute epidemic poliomyelitis, at which time the diagnosis of acute epidemic poliomyelitis was made on the other two cases occurring earlier in Dr. X.'s family.

Group II.

Five cases occurred in this group.

Case 4 occurred on May 24, 1910, and was attended by Dr. Z. No diagnosis was made. Case 17 occurred on June 14, in Dr. Z.'s family. Case 20 (possible abortive attack) occurred on June 15 in Dr. Z.'s family. Case 37 occurred on June 24, attended by Dr. Z. Case 133 occurred on August 21, and gave a history of contact with case 4, which occurred at a picnic, when they were swinging in a hammock together some time in July.

Group III.

Five cases occurred in this group.

Case 11 occurred on June 11, 1910, was taken to the Springfield Hospital and assigned to the care of a nurse. Case 13 occurred in a younger sister in the family of this nurse on June 12, 1910.

Cases 27 and 30 occurred respectively on the 20th and 21st of June, 1910, in a family which lived near the home of case 13. Case 13 was in intimate contact with cases 27 and 30, caring for them and playing with them.

Case 119 occurred on August 7 in a family in the same neighborhood, and gave a history of intimate contact with cases 13, 27 and 30.

Group IV.

Four cases occurred in this group.

Case 23 occurred in the five-year old daughter of Dr. XY. on June 18.

Case 39 occurred on June 28 in the family of a grocer clerk, who took orders from, and carried foodstuffs to, Dr. XY.'s family, and came into intimate contact with case 1.

Case 124 occurred on Aug. 11, 1910, after Dr. XY., father of case 23, had been in medical attendance for a week or so.

Case 127 occurred on Aug. 15 in a family where the father of case 39 was taking orders and delivering foodstuffs.

Group V.

Five cases occurred in this group.

Cases 8 and 14 occurred respectively on the 4th and 12th of June, 1910, in the family of a Greek fruiter, who traveled over a very extensive route in the city of Springfield with his fruit team.

Case 34 occurred on the 26th of June, 1910, and proved fatal, in a family

where the father of cases 8 and 14 sold fruit and came into contact with the children.

Case 48, an abortive attack, occurred on the 30th of June in the family of case 34.

Case 93 occurred on July 19 in an adult, which proved fatal. The Greek fruiter, father of cases 8 and 14, bought fruit at a wholesale warehouse, where he came in contact with the husband of case 93.

Group VI.

Four cases occurred in this group.

Case 72 occurred on July 12, a girl aged 11.

Case 82 occurred on July 14, a boy who lived opposite case 72, in which instance the contact was intimate.

Case 90 occurred in the twin brother of case 72 on July 16, contact intimate with cases 72 and 82.

Case 110 occurred in a girl at school, and gives a history of contact with cases 72, 82 and 90, and occurred in the same apartment house as case 82. Immediately after the sickness of case 82 the family moved out, and a week before the onset, July 31, in case 110, the family of case 110 moved upstairs into the apartments vacated by case 82.

Group VII.

Seven cases occurred in this group.

Case 18 occurred on June 14, 1910. Case 81 later occurred on July 14 in the same family. A dog was found sick and paralyzed, and shipped to Dr. Theobald Smith at the Harvard Medical School. The lesions in the cord of this animal were not suggestive of epidemic poliomyelitis. Cases 45 and 54 occurred respectively on June 29 and July 3, 1910, in the family neighboring to that of case 18, in which there were visiting and intimate contact between the children. A kitten in family of case 1 was fondled a good deal by all of the children in the family, especially by cases 45 and 54.

Case 57 developed in an adult on July 5, 1910, who previously visited the family of cases 18 and 81, and to whom was given the aforesaid kitten, which later became sick and developed a peculiar, staggering gait, and recovered.

Case 81, already referred to, occurred on July 14 in the same family as case 18.

Cases 128 and 128a occurred on August 16 and August 18 in the same family, the latter case being an abortive attack. There was intimate contact between these children and cases 18 and 81.

Group VIII.

Five cases occurred in this group.

Case 70 occurred on July 12.

Case 85 occurred on July 15, and both remained unrecognized and unreported for a month to six weeks.

Case 101 occurred on July 23 in the family of a mailman, who delivered mail to the family of cases 70 and 85.

Case 109 occurred on July 31, and gives a history of visiting in intimate contact with case 101.

Case 130 occurred in a young adult on August 18, and gave a history of visiting in a house next door to case 85, to which house case 85 had access, and with whom she occasionally came in contact.

Group IX.

Four cases occurred in this group.

Case 10 occurred on June 9 in a two-year old child. Two of his older sisters took care of the child during her sickness, and thus came into intimate contact with the sick child. They took part in a church play called "Little Women." The play was given on June 19, but rehearsals were performed twice weekly, commencing on the 19th of April and lasting until the time the play was given.

Case 12 developed on June 11 in a boy aged 12, whose sister likewise took part in the church play, and who himself attended the play and some of the rehearsals, and visited the home of case 10.

Case 41 occurred in a girl aged 11 on June 29, who took part in the church play, and came in intimate contact with the three sisters of cases 10 and 12, and visited their houses also.

Case 89 occurred on July 16. He took part in the same church play, and came in contact with case 41 and the sisters of cases 10 and 12. He also came in contact with case 12 at his home, visiting and playing with the latter.

Case 41 and the sisters of cases 10 and 12 were girls of an age (twelve years), and were in the same class in Sunday School.

Group X.

Four cases occurred in this group.

Case 36 gives a history of contact with case 29 in group IV; which latter case was reported two days later.

Case 3 occurred on the 8th of May, 1910. This case and case 21, which had its onset on June 16, were neither reported or isolated. They were traced when the third case (36) occurred on the 26th of June and was reported.

Case 52 occurred on the 2d of July.

These cases were all closely related by constant and intimate contact.

Group XI.

Four acute cases occurred in this group, with a history in connection with case 35 of a chronic case in the vicinity with which 35 was in constant contact.

Cases 25 and 26 occurred each on the 19th of June, 1910, in the family of a barber, who had placed in his front room a barber's chair and plied his trade there. There is also a history in connection with cases 25 and 26 of an

older sister, who eleven years previously had had a similar attack, and, a little before the onset of cases 25 and 26, gives a history of having had a slight gastro-intestinal upset.

Case 29 occurred on the 21st of June in a boy who lived opposite, and who had been in intimate contact with cases 25 and 26, and with a chronic case in the same family for several years, and had been recently in their house for the purpose of having his hair cut.

Case 35 occurred on June 26, and the history in this case was that the mother of case 29 and the mother of case 35 were cousins and were visiting each other's houses about this time.

Group XII.

Three cases occurred in this group, with a history of a line of contact between this group and case 31 in Group I. All these cases were in Jewish families.

Case 38 occurred on June 27, and case 12 on June 29. The history in these two cases was that the children for some time previous were in intimate contact.

Case 78 occurred on the 14th of July, and gives a history of visiting previously in the families of cases 38 and 42.

These groups could be multiplied both for Springfield and the surrounding neighborhood.

Detailed Analysis of Cases.

Out of the 254 cases thoroughly investigated by me in western Massachusetts, the details of 200 of the cases which occurred in Springfield, including a few which occurred in the immediate neighborhood of Springfield, are presented in the following analysis in tabular form.

**Analysis of Cases thoroughly studied (200 Cases in 186 Families).*[1]

Total number of children,		488
Number of recent illnesses,		253
Number of children paralyzed,	172	
Number of adults paralyzed,	28	
		200
Possible abortive cases,		53

The above table presents a very conservative estimate of possible abortive cases, numbering 53. It is a comparatively easy matter to find by careful inquiry that along with the more typical cases of acute epidemic poliomyelitis a number of cases occur that present a picture of a general

[1] The tables marked with an asterisk have already appeared in another paper by Lovett and Sheppard. Boston Medical and Surgical Journal, Vol. CLXV., No. 21, pp. 737-742, May 25, 1911.

infection with more or less nervous manifestations. These have not in all instances been included as probable abortive cases, and in the above table only those are included which were sufficiently severe to be regarded as border-line cases, and especially when they gave a history of contact, either directly with a paralytic case, or indirectly with a healthy third person.

Immediate Surroundings. — The tables that follow will present the conditions in the most concise manner, and wherever any significant point is presented comment will be made.

Nearness of House to Railroad.

	Cases.
On railroad,	11
Off railroad a block,	31
Within ⅛ mile of railroad,	11
Within ¼ mile of railroad,	31
Within ½ mile of railroad,	77
Within 1 mile of railroad,	39
	200

Bearing in mind that at least 150 of these cases were situated in Springfield proper, it is interesting to note that the majority of the cases are situated in close proximity to the railroad. And from the following table it will be seen that more than half of the cases were situated on the highroad.

Nearness of House to Highroad.

	Cases.
On the highroad,	116
Just off the highroad,	84
	200

At least half of the cases were on streets that were watered, and in a little less than half of the cases the streets had no treatment at all.

Treatment of Streets on which Cases occurred.

	Cases.
No treatment,	86
Watered,	97
Oiled,	9
Watered and oiled,	8
	200

Location of House.

	Cases.
Dry,	171
Damp,	29
	200

It is shown that at least 85 per cent. of the houses are dry, and these were for the most part situated on high ground.

The preceding tables, when taken with the table that follows, which speaks of the amount of dust which was estimated in every case, deficiency of rainfall and the increase in temperature, might be of interest.

Relation to Dust.

	Cases.
No dust,	–
Very little dust,	–
Moderate amount of dust,	112
Much dust,	81
Excessive amount of dust,	7
	200

A careful estimation was made of the dust in the immediate localities where cases occurred, and the table given above may be taken as fairly accurate.

Nearness to Water (Stream, Pond or Beach).

	Cases.
Off the water a block,	27
Within ⅛ mile,	18
Within ¼ mile,	71
Within ½ mile,	56
Within 1 mile,	28
	200

It is shown from the above table that more than half of the cases were situated within a quarter of a mile from the water, 56 within half a mile, and only 28 over half a mile from the water; the water in this case being for the most part the Connecticut River.

Age of House.

	Cases.
New house (1 to 10 years),	77
Old house (10 years and over),	123
	200

It is shown here, as was shown in the 1909 work, that more cases occurred in the older houses than in the new, and the above table may be taken as even more exact than that of 1909.

Sewage and Sanitary Data.

The sanitary conditions as tabulated in the tables that follow are by no means an exhaustive study of the water supply and sewage disposal of the city of Springfield. It is merely a short account of what exactly was found in the dwellings in which the cases occurred and is not compared with dwellings in the vicinity in which no cases occurred. This subject will have to be gone into more exhaustively before any definite conclusions can be arrived at.

Sanitary Conditions.

	Cases.
Excellent,	62
Good,	81
Fair,	42
Bad,	15
	200

Character of Sewage Disposal.

	Cases.
Sewer (city and town, mostly Springfield),	170
Cesspool,	10
Vault,	6
Privy,	14
	200

Character of Water Supply.

	Cases.
City,	151
Town,	31
Well,	13
Spring,	5
	200

When these tables are compared it will be seen that biting insects, vermin, etc., in families were present in greater number than history of bites; and I am inclined to the belief that the truth as to bites has been in a large measure withheld for obvious reasons.

Possible Favoring Conditions preceding or attending Infection.

	Cases.
Insect bite or sting,	61
Sore throat,	32
Diarrhœa,	28
Measles,	8
Wounds,	5
Whooping cough,	3
Sore eye,	3
Otitis media,	1
Had 1 or more of above possible sources of infection,	127
No such history,	73
	200

History of Insect Bites in 200 Cases.

	Cases.
No such history,	139
History of mosquito bites,	54
History of bedbug bites,	4
History of mosquito and bedbug bites,	2
History of bee sting,	1
	200

From the following table it will be seen that flies were prevalent in practically all of the cases, and biting insects present in a considerable proportion of the cases, as also rodents.

**Presence of Vermin, Insects, Rodents, etc., in 185 Families.*[1]

Flies were present in 185 families.
Mosquitoes were present in 67 families.
Ants were present in 62 families.
Mice (house) were present in 60 families.
Bedbugs were present in 39 families.
Roaches were present in 29 families.
Spiders were present in 26 families.
Rats were present in 23 families.
Biting flies were present in 7 families.
Field mice were present in 4 families.
Fleas were present in 2 families.
Bees were present in 2 families.
Sand fleas were present in 1 family.
Snakes were present in 1 family.
Crickets were present in 1 family.
Squirrels were present in 1 family.
Leeches were present in 1 family.

[1] See note, page 113.

In my study of the 1910 cases in and around Springfield I was repeatedly told that the "house fly" had been guilty of biting. There is no doubt that *Stomoxys calcitrans L.* was mistaken for the more common house fly (*Musca domestica L.*) In 1911, in the first and only case reported in Springfield up to September, I found *Stomoxys calcitrans L.* on the front door of the house, and the case (a young adult) gives a history of insect bites.

Paralysis in Domestic Animals. — The table shows that out of 186 families in which acute epidemic poliomyelitis occurred, 34 homes had illness, paralysis or death in 82 animals. One hundred and ten of the families above mentioned had animals, therefore about 30 per cent. of 110 families with animals had illness, paralysis or death in their animals.

Data as to Domestic Animals.

	Families.
No animal of any kind in	76
Animals present in	110
	186

27 homes had 31 dogs without sickness.
50 homes had 66 cats without sickness.
39 homes had about 745 hens and chickens without sickness.
11 homes had 18 horses without sickness.
3 homes had 3 cows without sickness.
4 homes had 4 canaries without sickness.
2 homes had 10 goldfish without sickness.
1 home had 5 geese without sickness.
1 home had 10 ducks without sickness.
1 home had 10 pigeons without sickness.
6 homes had 14 hens with sickness.
6 homes had 6 cats with sickness.
4 homes had 4 dogs with paralysis.
4 homes had 12 hens with paralysis.
2 homes had deaths in 2 dogs.
6 homes had deaths in 6 cats.
8 homes had deaths in 42 hens.
2 homes had deaths in 2 horses.
34 homes had illness, paralysis or death in 82 animals.

Nativity. — From these tables it will be seen that the colored race is not, as has been supposed, immune to the infection of acute epidemic poliomyelitis, since four cases occurred in the colored race in Springfield. Table A, giving the relative incidence per 1,000 in the colored

and white races for Springfield, shows that the colored race is considerably higher than the white.

	Cases.
American,	101
Irish American,	42
French,	19
Swedish,	11
Hebrew (Jewish),	8
Polish,	5
Colored,	4
Italian,	4
Scotch,	3
Greek,	2
Bohemian,	1
	200

Table A.

	Springfield Population.	Cases.	Incidence per 1,000.
White,	72,196	144	1.99
Colored,	1,294	4	3.09

When the question is considered for the whole State (Table B), it will be seen that 6 cases occurred in the colored race, and that the relative incidence in Massachusetts in 1910 per 1,000 of population was less in the colored race, being .17 and in the white .20.

Table B.

	Massachusetts Population.	Cases.	Incidence per 1,000.
White,	2,966,762	595	.20
Colored,	34,041	6	.17

Exposure to Heat, Cold or Dampness preceding the Attack.

	Cases.
To heat,	35
To cold,	7
To dampness,	30
Not exposed,	128
	[1] 200

[1] In 36 per cent. of the 200 cases.

Accident, Overexertion or Fall preceding the Attack.

	Cases.
Had no such history,	136
Had history of accident,	5
Had history of overexertion,	26
Had history of fall,	31
	[1]200

Swimming, Wading or Paddling.

	Cases.
Swimming, just before attack,	19
Wading, just before attack,	6
Paddling, just before attack,	7
No such history,	168
	[2]200

No relation to vaccination could be definitely traced in any of the cases, although in some cases a history of having been vaccinated a few weeks before onset was given.

Rabies has not been reported in the Springfield epidemic zone since 1908; two calves, however, are said to have died in West Springfield with symptoms resembling rabies in cattle. These calves are said to have been attacked by a dog that acted strangely and was destroyed. Landsteiner and Levaditi[3] observe that there is a striking analogy between poliomyelitis and rabies, as shown in (*a*) the progress of the virus along the nerve fibers, (*b*) affinity for the nerve centers, (*c*) reaction to physical and chemical agents, (*d*) filterability; but these investigators have shown that immunity to infantile paralysis does not give immunity to rabies. From the laboratory and field findings it is shown that the diseases are dissimilar.

Diseases Prevalent in Town at Time of Occurrence of Infantile Paralysis. — The diseases prevalent in the town at the time of the occurrence of infantile paralysis, with special reference to Springfield and the neighboring towns, were la grippe (influenza), summer diarrhœa, cases with symptoms of general infection, cases with gastro-intestinal disturbances, indefinite upsets among children with unusual nervous symptoms, and cases with typhoidal symptoms (negative Widals).

Communicability. — The question has been raised by Wickman as to whether or not the disease is transferable from case to case. Before

[1] In 31 per cent. of the 200 cases.
[2] In 16 per cent. of the 200 cases.
[3] Landsteiner and Levaditi: Annales de l'Institut Pasteur, Paris, Nov. 25, 1910.

1905, in which year he made an extensive study of the Swedish epidemic, Medin expressed the belief that contagion was possible but not probable, finding only one case in the epidemic in Stockholm in which he could give a clear history of contact.

Leegaard, also, in speaking of an epidemic which spread inland from the coast, gives the impression that the disease was communicable, but no proof is offered. Cordier and Platou, in their observations of epidemics of this disease, suggest contagion. Nannested, on the other hand, observed an epidemic in which he thought contagion was not probable.

We may say, then, that until 1905 no sure proof was offered as to the communicability of acute epidemic poliomyelitis. Since 1905, the date of the great epidemic in Sweden, in the classical work which was published by Wickman two years later, his experiences and the experiences of those who have later worked in the field, all go to show that acute epidemic poliomyelitis should be regarded as not only an infectious, but a communicable disease.

The following table does not differ very materially from that already given for the cases studied in 1909. The history of contact is vouched for under the various heads. There were many other indefinite histories of contact, but in order to make the table conservative and more sure, where there was any question of doubt in the minds of the patient or family, such cases were not included.

Instances of Contagiousness.

	Cases.
Certain direct contact with acute case,	32
Certain direct contact with possible abortive case,	4
Certain direct contact with chronic case,	1
Certain indirect contact with acute case by third person,	10
	47

There was intimate and constant contact in all these cases, hence no certain means of determining the day of the illness on which the sick child gave the disease to the other, or, in other words, the period of incubation in the recipient.

The table that follows is suggestive in so far that 14 families had more than 1 case in each, one of which had 3 cases. Were we justified in this stage of our knowledge in counting in the abortive cases, then the 172 families reported with 1 actual paralytic case in each, would be reduced, and the 14 families of more than 1 case would be increased proportionately.

Families with More than One Case.

Families with 1 case,	172
Families with 2 cases,	13
Family with 3 cases,	1
	186

As to the method of spreading the virus, or the manner, means or point of contact: from all that has been said in regard to the communicable nature of the disease, and from the impressions that one naturally received in the field, evidence is accumulating that points to personal contact, either with the sick during the prodromal, acute, or subacute stage, or with third persons not sick with the disease, but in attendance on the sick. This is further evidenced by the fact that six cases occurred in physicians' families, and one in the family of a nurse attending a case.

Paralysis in Family, Friends or Neighbors, before or since Attack.

	Cases.
No such history,	102
Of recent origin, that is, during 1910: —	
History of paralysis in family,	4
History of paralysis in friends,	29
History of paralysis in neighbors,	28
History of paralysis in family and friends,	5
History of paralysis in friends and neighbors,	25
History of paralysis in family, friends and neighbors,	4
Of remote origin, that is, chronic cases: —	
History of paralysis in family,	1
History of paralysis in neighbors,	1
History of paralysis in friends and neighbors,	1
	200

Diet.

	Cases.
General (including meat, fish, fruit, berries, cereals, bread, milk and eggs),	178
General without meat,	1
General without fish,	2
General without milk,	1
General without eggs,	1
Modified, bottle, bread and eggs,	12
Breast milk and bread,	1
Breast alone,	4
	200

Cases taking General and Modified Diet.

	Cases.
Meat,	185
Fish,	185
Fruit,	188
Berries,	108
Bread and crackers,	200
Eggs,	172
Vegetables,	185
Ice-cream cones,	5
Condensed milk,	1
Cereals,	125
Canned goods,	120
Ice cream,	75
Candy,	25
Raw cow's milk,	188
Breast milk and other food,	5
Breast milk alone,	4

In this series of 200 cases, 4 babies, all under six months, were said to be fed on breast milk alone. Many of the cases ate of several kinds of food.

The diet was gone into in detail since many cases were reported with gastro-intestinal symptoms, but from a careful study of the table given above, in which it is shown that many patients ate of a variety of food-stuffs, no definite conclusion can be drawn.

Habits and Amusements.

	Cases.
Digging in dirt (barn, yard, street),	116
Out-door life,	59
Creeping in house,	8
Athletics,	3
Barefooted,	2
Nursing babies,	4
Housewife,	2
Cleanly,	2
Working man (not cleanly),	1
Domestic,	1
Music teacher and athletic,	1
Addicted to the use of intoxicants,	1
	200

The above table shows that considerably more than half of the cases, which were amongst children, gave a history of digging in the dirt,

either in the barn or in their yard or in the streets. This, in connection with the out-door life of nearly a third of the cases, is interesting as showing the intimate relation between the cases and the soil.

An Estimate of Playmates.

	Cases.
In contact with at least 5 others, ...	82
In contact with at least 10 others, ...	86
In contact with at least 15 others, ...	7
In contact with at least 20 others, ...	22
In contact with at least 50 others, ...	3
.	[1]200

The above estimate is of the number of others not certainly known to have been sick, who either played with the patient, or, in the case of an adult patient, were more or less in contact before the onset of the illness.

The data are incomplete in so far that evidences of sickness developing in these groups of 5, 10, 15, 20 or 50 others, after contact with an actual case of acute epidemic poliomyelitis, are not available at present. In a few instances, however, where I have been able to look over the group of people who came in contact with the case, a secondary case of acute epidemic poliomyelitis has been traced. One very striking instance was that of a conductor, who during his prodromal stage, upon his car between Springfield and Feeding Hills, was in intimate contact with an adult aged forty-two, a storekeeper and barber at Feeding Hills. Within two weeks after the conductor became ill, the storekeeper was seized with an acute febrile onset of gastro-enteritis, and finally developed paralysis in both legs. (His wife had a similar attack with malaise, nausea, vomiting and diarrhœa, but developed no paralysis. Their only child, however, a boy of twelve years, was perfectly well during this time.) This storekeeper later furnished a probable indirect source of infection to the only other case in the little hamlet of Feeding Hills, in this manner: the father of a little child, two years of age, worked for the storekeeper and was shaved by him twice a week. Within a fortnight of the storekeeper's onset the little girl became sick and developed paralysis of the lower extremities. (Probable contact by healthy third person.)

[1] These cases later developed acute epidemic poliomyelitis.

Movements of Patient and Family previous to Illness (Trolley Rides, Trips to Beaches, Picnics).

	Cases.
Stayed around home,	7
Took trolley rides,	165
Took trolley rides and trips to beaches,	1
Took trolley rides and trips to beaches and picnics,	14
Took trolley rides and picnics,	13
	200

From this table it is seen that the majority of the cases up to the time of their acute illness were circulating in the community, and using public conveyances.

Incidence of the Disease by Sex.

	Cases.
Males,	112
Females,	88
	200

The charts that follow, giving the cases by age periods, show the greatest incidence of disease in children under five years, and for any one year, three years of age shows the largest number of cases. This table is given for 601 cases, on which reports were made during 1910. Another interesting thing brought out in this table is the greater frequency of the disease in young adults and amongst the aged. The youngest case was three months, the oldest eighty years of age.

By Age Periods.

	Cases.	Per Cent.
From birth to 12 months, inclusive,	51	8.6
From 13 months to 23 months, inclusive,	66	11.0
2 years old,	61	10.1
3 years old,	98	16.1
4 years old,	69	11.3
5 years old,	51	8.5
	396	65.5
6 to 10 years, inclusive,	93	15.3
	489	80.8
11–20 years, inclusive,	69	11.3
	558	92.1
21–30 years, inclusive,	28	4.7
	586	–
31–80 years, inclusive,	15	2.5
	601	–

Mortality by Age. — The average mortality by age as shown in the following table for 601 cases, is 10.31 per cent., an increase of 2.31 per cent. over that of 1909.

Table C.

	Cases.	Deaths.	Mortality (Per Cent.).
1 year,	117	11	9.40
2–10 years,	372	37	9.94
Over 10 years,	112	14	12.50
Totals,	601	62	–
Average mortality,			10.31

The mortality taken in three age periods, under one year, from one to ten, and over ten, shows that the greatest mortality per hundred was in the group of cases over ten years of age, which was 12.5 per cent.

The mortality by age has been worked out in shorter age periods in Table D, which repeats the data given in Table C in more detail, and shows the highest mortality in children under five years of age.

Table D.

Age.	Cases.	Deaths.	Mortality (Per Cent.).
From birth to 12 months, inclusive,	117	11	9.40
2 years,	61	8	13.11
3 years,	98	10	10.20
4 years,	69	10	14.49
5 years,	51	2	3.90
6–10 years, inclusive,	93	7	7.52
11–20 years, inclusive,	69	7	10.14
21–30 years, inclusive,	28	5	17.80
40 years and over,	15	2	13.33
Totals,	601	62	–
Average mortality,	–	–	10.31

Skin Eruption. — Nine cases out of 200 gave a history of some skin eruption appearing on the chest or around the neck or about the girdle. In one or two such cases I have noticed a small maculopapular rash with a faint blush underlying the affected area.

Suspicious contemporary sickness in Springfield and in neighboring towns was frequently heard of. One such little epidemic was investi-

gated in Russell. Another such group of cases of influenza-like attacks occurred in Ashfield.

These cases all gave more or less a picture of the "abortive" type of acute epidemic poliomyelitis as spoken of by Wickman.

In Springfield after the epidemic of 1910 several doctors said that they had observed a number of obscure gastro-intestinal and rheumatic cases, in which a positive diagnosis was not made. In a retrospect all are inclined to regard these cases as possibly poliomyelitis.

Urine. — The reports show that out of 200 cases the urine was examined in 15 cases and found to be negative in 14 cases. In 1 case a trace of albumen was found, and 1 instance was reported where a clear green urine was voided.

Condition of Patient and Others in Family.

	Condition just before Attack.	Acute Illness within Four Weeks of Attack.	Recent Illness in Other Members of Family.
Perfectly well,	73	131	-
Malaise,	57	3	1
Upper respiratory passage troubles,	16	12	21
Acute gastro-enteritis,	9	10	15
Vomiting,	8	7	1
Pains and aches,	7	1	-
Excited — irritable,	7	-	-
Measles,	5	8	13
Tonsilitis,	2	4	3
Influenza,	1	2	1
Whooping cough,	1	2	5
Teething,	2	3	-
Paralysis of bladder,	1	-	-
Aphonia,	1	-	-
Vertigo,	1	1	-
Girdle heat,	1	-	-
Chill,	1	1	-
Cystitis,	1	1	-
Sore eye,	1	2	1
Abscess,	1	-	-
Punctured wound,	1	1	-
Operation,	1	3	-
Glands in neck,	2	2	1
Scarlet fever,	-	1	1

Condition of Patient and Others in Family — Concluded.

	Condition just before Attack.	Acute illness within Four Weeks of Attack.	Recent Illness in Other Members of Family.
Pneumonia,	-	1	1
Diarrhœa,	-	2	-
Otitis media,	-	1	-
Skin eruption,	-	1	-
Diphtheria,	-	-	4
Cholera infantum,	-	-	3
Tuberculosis,	-	-	3
Appendicitis,	-	-	1
Rheumatism,	-	-	1
Cancer,	-	-	1
Mental disturbance,	-	-	1
	200	200	-

The table shows malaise, upper respiratory passage trouble and gastro-enteritis to have been present more often than any other combination of ailments.

General Features of Acute Attack — Symptoms.

198 cases give history of fever.
184 cases give history of pain and tenderness.
117 cases give history of brain symptoms.
106 cases give history of headache.
79 cases give history of retraction.
59 cases give history of sore throat.
24 cases give history of apathy.
19 cases give history of delirium.
18 cases give history of rigidity of neck.
13 cases give history of cough.
13 cases give history of irritableness.
11 cases give history of restlessness.
11 cases give history of unconsciousness.
9 cases give history of tired condition.
8 cases give history of rigidity of spine.
8 cases give history of lassitude.
7 cases give history of strabismus.
6 cases give history of change of temperament.
6 cases give history of diaphragmatic breathing.
6 cases give history of dysphagia.

5 cases give history of sweating.
5 cases give history of irregular pulse and respiration.
5 cases give history of twitchings.
5 cases give history of diminished or absent reflexes.
4 cases give history of convulsions.
4 cases give history of nystagmus.
3 cases give history of weakness.
3 cases give history of hyperæsthesia.
3 cases give history of stupor.
3 cases give history of exaggerated reflexes.
3 cases give history of regurgitation of food.
3 cases give history of anorexia.
1 case gives history of thirst.
1 case gives history of anxious expression.
1 case gives history of tremor.
1 case gives history of coma.
1 case gives history of tympanites.
1 case gives history of insomnia.
1 case gives history of hiccough.

Data as to Lodgers.

	Families.
No lodgers in	176
18 lodgers in	10
	186

Details of Digestive Disturbance connected with Attack.

	Preceding Attack.	Accompanying Attack.	Following Attack.
Nausea and Vomiting,	83	39	1
Constipation,	78	94	85
Colic,	3	7	–
Diarrhœa,	27	28	8

Twenty-two had no digestive disturbance.

The striking feature in the above table is the fact that constipation is found preceding, accompanying and following the attack in nearly 50 per cent. of the cases.

Disturbance of Intestines during Attack.

	Cases.
No disturbance in,	75
Constipation in,	98
Diarrhœa in,	15

	Cases.
Involuntary defecation,	2
Constipation, later diarrhœa,	1
Diarrhœa, later constipation,	8
Constipation and colic,	1
	200

Disturbance of Bladder during Attack.

	Cases.
No disturbance in,	145
Retention,	39
Retention, later involuntary,	3
Incontinence,	4
Frequent,	5
Painful,	2
Involuntary,	1
Incontinence, later retention,	1
	200

Pain and tenderness are as striking features in the 1910 cases as they were shown to be in 1909.

Pain and Tenderness.

	Cases.
Pain or tenderness was present in	184
Pain or tenderness was absent in	16
	200

Pain or tenderness lasted: —	Cases.
No pain,	16
1 day or less,	4
2 days,	6
3 days,	7
4 days,	4
5 days,	4
1 week,	12
1–2 weeks,	16
2–3 weeks,	10
3–4 weeks,	7
4–5 weeks,	1
6–7 weeks,	1
A few days,	15
Until death,	28
Present when report was made,	69
	200

I have given the following extended table of the distribution of paralysis, which shows that many combinations of paralysis are met with, and that the abdominal muscles and muscles of respiration and deglutition are more often affected than has been hitherto supposed.

Distribution of Paralysis.	Cases.
One leg only,	34
Both legs only,	33
One arm only,	15
Both arms only,	4
One arm and one leg same side only,	7
One arm and leg opposite sides only,	4
Both legs and one arm only,	6
Both arms and one leg only,	1
Both arms and both legs only,	3
Ataxia (transitory),	5
Back only,	0
Abdomen only,	0
Neck only,	4
Respiration only,	5
Deglutition,	3
Intercostal only,	1
Face only,	0
Right face only,	5
Left face only,	5
Unsteady gait,	3
One leg and respiration,	3
One leg and neck,	1
One leg, one arm, abdomen, right face and respiration,	1
One leg, respiration and half face,	1
One leg and abdomen,	2
One leg and back,	3
One leg, back and abdomen,	1
Both legs and respiration,	2
Both legs, back and neck,	1
Both legs, respiration and deglutition,	1
Both legs and back,	10
Both legs, abdomen and respiration,	1
Both legs and abdomen,	1
Both legs, back and abdomen,	1
One arm, both legs and back,	3
One arm and right face,	3
One arm, half face and deglutition,	1
One arm, both legs and respiration,	2
One arm, back, respiration, neck and abdomen,	1
Both arms and respiration,	1

	Cases.
Both arms, back, neck and respiration,	1
Both arms, back and neck,	1
Both arms, both legs, back and respiration,	5
Both arms, both legs, half face,	1
Both arms, both legs, back and abdomen,	2
Both arms, both legs, abdomen, back and respiration,	1
Both arms, both legs and respiration,	2
Both arms, both legs and back,	3
Both arms, both legs, respiration and half face,	1
Respiration and neck,	1
Respiration and deglutition,	1
Respiration and face,	1
Half face and deglutition,	1
Half face and neck,	1
	200

Appearance of Paralysis in Days and Weeks after Onset of Fever.

	Cases.
Same day,	20
1 day,	31
2 days,	40
3 days,	34
4 days,	15
5 days,	11
6 days,	11
7 days,	14
8 days,	4
9 days,	2
10 days,	2
11 days,	2
12 days,	4
13 days,	1
14 days,	1
2–3 weeks,	5
3–4 weeks,	1
4–5 weeks,	1
8 weeks,	1
	200

Treatment. — Cases receiving symptomatic treatment behaved much as in former years, the treatment, in general, seeming to have no specific effect upon the infection.

The Withdrawal of the Cerebro-spinal Fluid by Means of Lumbar Puncture. — The technique of this procedure is well-known, and I have

resorted to it in cases where it seemed to be indicated. It has proved a wise and safe procedure in diseases such as meningitis (tubercular and diplococcal), and is applicable to any disease of the nervous system where symptoms of cortical pressure are evident. A trial of this procedure in cases of acute epidemic poliomyelitis makes it appear that some good has been effected by it. In any case it has been proved useful both for its diagnostic and for its therapeutic value.

Possible Recurrent or Second Attacks of Acute Epidemic Poliomyelitis. — In September, 1910, Eshner reported a possible second attack of acute anterior poliomyelitis in the same patient. A review of this case is given in the "Journal of the American Medical Association," Oct. 8, 1910, page 1319, as follows: —

On a warm day in August, 1891, when twenty-five months old, after an indiscretion in diet, and without antecedent traumatism, Eshner's patient was seized with fever lasting three days, and associated with pain in the right leg and the back. There had been no vomiting and no diarrhœa. On the fifth day the right lower extremity was found to be paralyzed, without apparent alteration in sensibility. The paralysis increased in severity for a week, and then it began gradually to diminish. The upper extremities, the left lower extremity, and the face were unaffected. The general nutrition was preserved, but the right lower extremity was moderately wasted. Intelligence was good and sensibility was unaffected. The gait was wobbling, the feet being held rather far apart in walking. There were no contractures and no deformity. The knee-jerk was normal on the left, enfeebled on the right. The circumference of the right leg was 7¼ inches, that of the left 7½ inches. The muscles of the right leg responded less well to faradic stimulation than did the muscles of the left leg, but there was no degenerative reaction. The patient occasionally had nocturnal enuresis, but there was no evidence of rachitis. Under treatment with massage and electricity for eight months practical recovery took place. Eleven years after this illness, in March, 1903, a day after a fall, resulting in injury to the left shoulder and the left elbow, the patient developed weakness in both hands, more marked on the left. She had not been feeling well at this time and was "nervous," although she was attending school and had no fever or nausea or vomiting. While the symptoms manifested were those of acute anterior poliomyelitis, Eshner points out that some one might attribute them to peripheral nerve injury in consequence of the fall. The development of the symptoms a day following and not immediately after the accident, the involvement of both hands primarily, even though in slight degree and but transitory in character on the uninjured side, the absence of sensory alterations, certainly entitle the possibility of a spinal, rather than a peripheral, lesion — a poliomyelitis rather than neuritis — to serious consideration.

The symptoms of the first attack in Eshner's case are sufficiently well marked to be regarded as those of poliomyelitis.

Eckert, in the following quotation, gives a case of recrudescence: "H. Sch. was taken sick September, 1903, with poliomyelitis, and was paralyzed in the left leg. Tenotomy was done and splints applied. At the beginning of April, 1909, occurred a second infection, and paralysis of the right leg." [1]

A positive case of this nature has come under my observation. In a family living in a typically rural part of Massachusetts sixteen years ago a girl, then three years of age, had an attack of poliomyelitis, and recovered with a residual paralysis of the deltoid of the left arm. The family numbered, in 1910, 5 children — 4 girls and 1 boy. In 1910 a girl twelve years of age became violently sick and died in a few days. The attack was thought to have been ptomaine poisoning, though characterized by many nervous symptoms. Early in August, 1911, the family went for a vacation to Maine. After three weeks' stay in Maine a six-year-old girl was taken sick on August 24 and died with respiratory paralysis in four days. No diagnosis was made by the attending physician.

On August 28 the boy, three years and ten months old, was taken sick at Warren, Me., in a similar manner. There was, however, a slight paralysis of left face. The family then returned to Massachusetts, where the funeral of the girl was held.

On August 31 the oldest girl, nineteen years old (who when she was three years of age had poliomyelitis), was taken sick and within a week developed a distinct Landry's paralysis, involving all four extremities, neck, back, abdomen and respiration.

On September 2, a younger sister, aged sixteen years and nine months, was taken similarly sick. She developed rapidly the Landry type of poliomyelitis and died in a few days.

This group of cases is doubly interesting from the fact that 4 cases occurred in the same family, and further, that one of them, the nineteen-year-old girl, had had a previous attack of the same infection sixteen years before.

Prognosis. — Six months after the beginning of the epidemic outbreak in western Massachusetts, 27 cases out of 200 had completely recovered, that is, 13.5 per cent.

(*a*) *Analysis.*

	Cases.
Recovered,	27
Not yet recovered,	139
Deaths,	34
	200

[1] Eckert; Ueber das akute Stadium der epidemischen Kinderlähmung, nebst Bekanntgabe einer Falles von Poliomyelitis fulminans, Deut. Med. Woch., Jan. 19, 1911.

Of the 139 "not yet recovered," at least ⅕ (or about 30) are recovering, that is, the paralysis in these cases may be said to be disappearing.

Those cases reported to me as completely recovered I immediately subjected to a rigid and careful special examination along the lines indicated below, before they were counted in as recovered cases: —

1. Dorsal and plantar flexion of foot.
2. Inversion and eversion.
3. Knee extension.
4. Lying on back: —
   (*a*) Flexion on thigh with knee extended.
   (*b*) Adduction of leg.
5. Lying on side: —
   (*a*) Abduction of leg.
   (*b*) Rising from horizontal position to sitting.
6. Inspection of spine.
7. Raising arms above head.
8. Evidence of limp.
9. Evidence of flat foot.

(*b*) *Age.*

	Cases.
1–2 years,	2
2–3 years,	6
3–4 years,	2
5–6 years,	7
8 years old,	1
9 years old,	1
10 years old,	1
11 years old,	2
12 years old,	1
25 years old,	1
26 years old,	1
31 years old,	1
36 years old,	1
	27

(*c*) Recovery in these 27 cases occurred within eight weeks after initial symptoms of acute attack.

(*d*) The majority of the cases were moderately severe.

(*e*) The evidence of paralysis is vouched for both by the physicians in charge of the cases and by myself.

(*f*) *Extent of Paralysis in Recovered Cases.*

	Cases.
Two legs only,	5
One leg only,	4
Ataxia,	4
One arm and leg, opposite sides,	2
One arm only,	2
Neck muscles,	2
One arm and leg, same side,	1
One arm, both legs,	1
Unsteady gait,	1
Left face only,	1
Respiration,	1
Intercostal muscles,	1
Deglutition,	1
Bladder and ataxia,	1
	27

(*g*) *Duration of Paralysis.*

	Cases.
A few days,	4
About 1 week,	3
About 2 weeks,	8
About 3 weeks,	7
About 4 weeks,	3
About 6 weeks,	1
About 7 weeks,	1
	27

(*h*) *Pain and Tenderness in Recovered Cases.*

	Cases.
No pain or tenderness,	6
2 days,	1
3 days,	1
4 days,	1
A few days,	4
1 week,	1
1–2 weeks,	5
2–3 weeks,	2
3–4 weeks,	1
Present when report was made,	5
	27

(*i*) *Present Condition.* — Twenty-seven all well and healthy.

Personal Hygiene. — During my investigations I have been careful to observe the following precautionary measures, and have tried to inculcate the same in the families afflicted: —

(*a*) Washing of hands with soap and water before and after handling patients, excretions, etc.

(*b*) After handling patient and washing the hands, further rinsing in bichloride of mercury or equal parts of boracic acid and chloride of lime.

(*c*) Final rinsing in alcohol 70 per cent.

The following measures were carried out as often as practicable, especially before mixing in the company of healthy people: —

(*d*) Antiseptic mouth-wash and gargle with hydrogen peroxide or glycothymolin.

(*e*) Eye douche with boracic acid.

(*f*) Nasal spray with menthol preparation.

Finally, after seeing the last case for the day, all clothes worn and other articles, including the bag in which they were carried, were placed in a sealed cupboard and exposed over night to the influence of formaldehyde gas.

Conclusions.

Certain conclusions are evident from the tables given in the body of the paper; other conclusions worthy of especial emphasis are as follows: —

1. By far the largest focus of epidemic poliomyelitis occurring in Massachusetts in 1910 affected the city of Springfield.

2. From Springfield as a focus the disease seemed to spread radially, and there was a suggestion that its spread was due to infectious contact occurring in said city at various public gatherings.

3. Furthermore, the time relation of other cases occurring in the Connecticut valley seemed to make Springfield the source of these cases.

4. Although personal contact has been emphasized as being of especial importance in the spread of this disease, transfer through the mediation of biting insects might accord equally well if not better with the known facts.

5. The occurrence of second attacks of infantile paralysis after intervals of months or even years is very probable. The agency of such affected individuals as possible carriers of the disease in the intervals between their attacks is worthy of thorough investigation.

I take this opportunity of expressing my thanks to physicians in Massachusetts, whose cases I have investigated, for the help they have afforded me. Especially am I indebted to Dr. J. V. W. Boyd, State Inspector of Health (District 12), and Captain Young, agent of the Springfield board of health, who have assisted me in this work in every possible way.

THE POSSIBLE ETIOLOGICAL RELATION OF CERTAIN BITING INSECTS TO THE SPREAD OF ACUTE EPIDEMIC POLIOMYELITIS.[1]

By Charles T. Brues, Instructor in Economic Entomology, Harvard University, and Philip A. E. Sheppard, M.D., Boston, Mass.

In attempting to discover any connection between insects and the spread of epidemic anterior poliomyelitis, a number of facts must be considered. Much that has already been learned from clinical and epidemiological investigations can be turned to account, not only to reduce the number of insects which might be suspected of transmitting this disease, but also to suggest certain *a priori* conclusions to be tested out, both in the field and by experimentation.

It will be well to outline briefly the data which suggest insects as carriers, as well as to enumerate such facts as appear significant from an entomological standpoint.

1. The sporadic occurrence of the cases: this is not easily explained on the basis of ordinary contact infection, at least in many instances.

2. The seasonal distribution of the disease shows the largest incidence during the warmer months, when insects of all sorts are most prevalent.

3. The failure on the part of investigators to show that the disease spreads most where many children are regularly in close contact. It seems, on the contrary, from reports thus far published, to be the exception rather than the rule for several in a family to contract the disease, even though the number of young children be large.

4. The comparative immunity from this disease of large cities in which the proportion of biting insects to the human population must be less than in more sparsely settled or in rural districts, while the opportunities for personal contact are at a maximum.

5. The fact that in the towns of Massachusetts, where the disease has been most prevalent, the proportion of animals to the population is greater than in the cities, where the disease is least prevalent, in itself implies a larger number of biting and parasitic insects which may affect man as well as animals. Furthermore, when we consider the paralyses of animals and their possible relation to human anterior poliomyelitis, the study of biting parasites becomes increasingly interesting and important.

The following table shows that during the period July 30–Sept. 26,

[1] Abstract of a report submitted to the State Board of Health on Sept. 25, 1911.

1911, the homes of 88 patients in 17 cities and towns were investigated as to the presence of biting flies and insects in close proximity to the affected individuals: —

Cities and Towns visited.

Date of First Visit.	City or Town.	Number of Cases.
July 30,	Waltham,	2
Aug. 13,	Tewksbury,	6
Aug. 15,	Winthrop,	3
Aug. 17,	Woburn,	14
Aug. 24,	Somerville,	10
Aug. 26,	Newton,	4
Aug. 30,	Winchester,	2
Aug. 31,	Pocasset (Bourne),	2
Sept. 6,	Hamilton,	1
Sept. 6,	Marblehead,	1
Sept. 7,	Marlborough,	6
Sept. 16,	Lowell,	14
Sept. 16,	Westford,	1
Sept. 20,	Roxbury,	4
Sept. 25,	Winchendon,	1
Sept. 26,	Fall River,	13
Sept. 26,	New Bedford,	4

Stomoxys calcitrans L., the ordinary biting stable fly, was the only biting insect other than mosquitoes to be found constantly in the immediate vicinity of the patients. Interesting facts bearing upon this aspect of the problem are as follows: —

In Woburn, where 14 cases occurred, a paralytic affection of pigs was reported previous to the beginnings of the human disease. In another instance a cat was paralyzed.

In Lowell, furthermore, in one family a cat became paralyzed coincidently with a child in the same family. Moreover, the cases in Lowell were distributed along the main artery of traffic from the city of Woburn, and situated either on or sufficiently near the main thoroughfare to be within easy flying distance of it. In other words, it is possible that infected flies from Woburn may have followed horses or may have been carried on vehicles from Woburn into Lowell. In point of time Woburn cases occurred first.

In Somerville one case gave the history that, one month before the

attack, the patient was stung between the shoulders by a strange insect, and at the same time an adult member of the same family was bitten in the same way. The sting was accompanied by a sharp pain and was probably due to some species of *Tabanus,* although it may have been that of *Stomoxys* or other biting insect. In two instances in Somerville the fathers of the affected children were teamsters, and in one instance two older boys in the family were also teamsters. The implication is, of course, that teamsters, being in intimate contact with horses, might bring home with them infected biting flies. Furthermore, another home was situated in a narrow court close to the freight yard of the Boston & Maine railroad. Wickman and others have noted a close relation between anterior poliomyelitis and railroad tracks. It is suggested that the transportation of food-producing animals over the railroads and the constant association with these animals of biting flies, may have an important bearing on the occurrence of this disease. One victim in Attleborough was a freight brakeman.

In Newton one case occurred on the outskirts of the city, where several cows were reported to have been lame in the hind quarters two weeks before the child was taken sick. The cows were pastured in a field adjoining the house where the patient lived, and frequently escaped into the back yard of this house.

In a case which occurred in Hamilton the father was a coachman, and thus naturally had much to do with horses and other animals.

In Fall River, an adult, male, seventy-three years old, gave, unsolicited, a history of bites by stable flies a week or ten days previous to a febrile attack and paralysis of one arm.

Summary.

Nothing absolutely definite has hitherto been ascertained regarding the channels of infection of acute epidemic poliomyelitis.

Many facts connected with the distribution of cases and the spread of epidemics of this disease, together with histories of insect bites, suggest at least that the disease may be insect-borne.

From our field work during the present summer, together with a consideration of the epidemiology of the disease, it is suggested that *Stomoxys calcitrans L.* may be responsible for the spread of acute epidemic poliomyelitis. No facts which disprove such hypothesis have as yet been adduced.

Experiments based upon this hypothesis are now in progress.

AN INVESTIGATION OF THE BLOOD IN CASES OF ACUTE EPIDEMIC POLIOMYELITIS (INFANTILE PARALYSIS).

By J. W. Hammond, Jr., A.B. (Fourth-year Student, Harvard Medical School), and Philip A. E. Sheppard, M.D., of Boston.

The report that follows (submitted Nov. 15, 1911) is a preliminary attempt to determine the blood picture in cases of acute epidemic poliomyelitis (infantile paralysis), and especially in the prodromal stage of the disease.

Lucas [1] in his work on monkeys has shown a leucopenia with a lymphocytosis in the prodromal stage of infantile paralysis.

The plan was to work in an epidemic field, if one could be found, and to make white counts and differential counts of the leucocytes from (*a*) actual cases of the disease, (*b*) suspicious cases, and (*c*) from individuals exposed to infection but well. An extensive epidemic did not occur, however. All the cases in which blood films were obtained were scattered, sporadic cases in the immediate vicinity of which no secondary cases were discovered. Two small epidemics, unfortunately, did not afford us any material in the prodromal stage.

The blood counts made from those exposed to the sporadic cases proved negative in every way. The more important part of this work is, therefore, incomplete.

The following data, presented in tabular form, give the epidemiological and clinical details of 13 cases, in 2 of which blood examinations were made one day before paralysis, and in 1 of which the blood examination was made practically coincident with the appearance of paralysis: —

[1] Lucas, W. P.; The Diagnosis of Infantile Paralysis in the Prodromal and Early Acute Stage, in "Monthly Bulletin" of the Massachusetts State Board of Health, June, 1910.

Before Paralysis appeared.

Number of Case.	Case.	Epidemiological and Clinical Data.					Blood Picture.						
		Condition before Onset.	Date of Onset.	Character of Onset.	Date of Paralysis.	Extent of Paralysis.	Condition of Patient when Blood taken.	Total White Count.	Poly. (Per Cent.).	Lymph. (Per Cent.).	Trans. (Per Cent.).	Baso. (Per Cent.).	Eosin. (Per Cent.).
16a, . .	H. C., male, three years.	Constipated, but in good health.	Aug. 17, 1911.	Gastro-intestinal upset, with pain along spine.	Aug. 20, 1911.	Right arm.	One day before paralysis; was hyperæsthetic and restless.	18,000	72	26	2	–	–
19, . .	H. B., Male, two years.	Irritable for one week, feverish and hyperæsthetic.	Aug. 13, 1911.	Febrile, irritable. General pains.	Aug. 22, 1911.	Extensors of left leg.	One day before paralysis; was irritable and hyperæsthetic.	15,000	80	20	–	–	–

Coincident with Paralysis.

4, . .	B. C., female, two years.	Eczema of scalp; pale and apathetic.	July 28, 1911.	Febrile.	July 30, 1911.	Right leg, left face.	Seen four hours after left facial paralysis; right K. J. gone; apathetic.	17,200	65	29	5	–	1

In the tables that follow the blood counts are given of cases examined at different periods, from one day to fourteen days, after paralysis appeared.

One Day Post Paralysis.

Number.	Age (Years).	Count.	Poly. (Per Cent.).	Lymph. (Per Cent.).	Trans. (Per Cent.).	Baso. (Per Cent.).	Eosin. (Per Cent.).	Remarks.
7, . .	3	13,900	50	50	–	–	–	– –
26, . .	5	15,000	55	45	–	–	–	– –

Two Days Post Paralysis.

23, . .	2	12,000	50	38	7	2	3	– –
32, . .	3	18,500	60	40	–	–	–	– –

Three Days Post Paralysis.

9, . .	2	14,500	60	40	–	–	–	– –
12, . .	3	22,000	51	35	14	–	–	– –
30, . .	22 [1]	18,000	33	57	8	–	–	Cervical adenitis of one year's standing.

Four Days Post Paralysis.

13, . .	4	18,000	60	38	2	–	–	– –
24, . .	6½	12,000	72	26	2	–	–	– –
15, . .	7	12,000	68	30	–	1	1	– –

Five Days Post Paralysis.

18, . .	7	16,200	73	27	–	–	–	– –
20, . .	7	12,200	64	28	–	5	3	– –
21, . .	4	16,000	50	44	6	–	–	– –
22, . .	15	9,800	70	25	5	–	–	– –
25, . .	22 [1]	14,000	60	40	–	–	–	– –
37, . .	12	13,000	70	30	–	–	–	– –
4, . .	–	11,400	60	22	13	2	3	Second visit.

Six Days Post Paralysis.

14, . .	1	11,300	69	25	1	–	5	– –
29, . .	2	18,000	62	33	5	–	–	– –

[1] Months.

Seven Days Post Paralysis.

Number.	Age (Years).	Count.	Poly. (Per Cent.).	Lymph. (Per Cent.).	Trans. (Per Cent.).	Baso. (Per Cent.).	Eosin. (Per Cent.).	Remarks.
26a, . .	5	14,900	60	40	–	–	–	– –
8, . .	5	14,900	77	23	–	–	–	– –
28, . .	1	9,000	40	60	–	–	–	– –
34, . .	2	10,000	49	48	3	–	–	– –
35, . .	7	9,600	62	36	2	–	–	– –
31, . .	6	24,000	–[1]	–[1]	–[1]	–	–	– –
3, . .	–	6,600	38	58	4	1	–	– –

Eight Days Post Paralysis.

26a, . .	5	11,300	65	35	–	–	–	– –

Nine Days Post Paralysis.

38, . .	–	–	60	38	2	–	–	–. –

Ten Days Post Paralysis.

11, . .	1½	18,200	60	33	6	1	–	– –
27, . .	7	8,000	45	51	4	–	–	– –

Thirteen Days Post Paralysis.

6, . .	–	23,400	22	73	5	–	–	– –

Fourteen Days Post Paralysis.

5, . .	6[2]	10,200	25	58	17	–	–	– –
15, . .	–	8,100	53	45	3	–	–	– –
26a, . .	5	19,900	77	23	–	–	–	– –

[1] Polynuclears in majority. [2] Months.

Summary.

1. In two instances in the preparalytic stage of anterior poliomyelitis a slight but definite polymorphonuclear leucocytosis was observed.

2. A slight polymorphonuclear leucocytosis was present in one case coincident with the definite onset of paralysis.

3. A subsidence of this leucocytosis occurred in the majority of cases within four days subsequent to the onset of paralysis, but in others the leucocytosis persisted as long as two weeks.

EXPERIMENTS AS TO THE PROTECTIVE VALUE OF CERTAIN SPECIFIC SERA AND VACCINES AGAINST THE VIRUS OF POLIOMYELITIS.

By William P. Lucas, M.D., Boston, and Robert B. Osgood, M.D., Boston.

The following experiments were undertaken in December, 1911, at the request of the Massachusetts State Board of Health, with the purpose of ascertaining whether an immunity to various bacterial infections, artificially produced in monkeys by means of repeated and graded injections of vaccines and sera, produced, also, an immunity or partial immunity to the virus of poliomyelitis. The experiments were carried out in the laboratory of surgical research of the Harvard Medical School.

Our thanks are due to Dr. H. C. Low for performing the autopsies and making the microscopic examinations which form the basis of the autopsy reports.

While these experiments are not large in number, the findings are so definite as to be almost conclusive, and suggest strongly the probability that an immunity to the commoner bacterial infections in no way protects the human race from anterior poliomyelitis.

The monkeys used were apparently healthy untamed *Rhœsus*. The virus was a very active strain obtained from the Rockefeller Institute, through the kindness of Drs. Flexner and Clark, and in every case was injected intracerebrally. Sterile salt solution emulsions of the spinal cord which had been preserved in 50 per cent. glycerine and .5 per cent. carbolic solution were used, and in no case, either during the days immediately following the inoculation nor at autopsy, were there any signs of pyogenic infection at or near the inoculation wound. The small skin wounds healed in every case by first intention.

Monkey No. 106 was immunized with anti-dysenteric serum, obtained from Mulford and Company for experimental purposes. Three injections of 10 cubic centimeters each were given on December 3, December 8 and December 15 intraperitoneally. On December 18 he received 2 cubic centimeters of the cord emulsion from Flexner and Clark's series M. A. intracranially. First symptoms were noted December 22, five days after the inoculation. Progressive paralysis ensued, followed by death December 24. Autopsy typical of poliomyelitis. See autopsy protocol.

Monkey No. 107 received three injections with anti-streptococcus serum obtained from the Parke, Davis experimental laboratories. It was a polyvalent serum. The injections were given intraperitoneally in 10 cubic centimeter doses on December 3, December 8 and December 15. On December 18 he also received 2 cubic centimeters of the same cord M. A. emulsions. First symptoms appeared December 22, complete paralysis and death by the 26th. Autopsy typical of poliomyelitis.

Two control monkeys, Nos. 117 and 118, were inoculated on December 18, receiving 2 cubic centimeters of the same cord M. A. emulsion intracranially. Both control monkeys showed symptoms within a few hours of the treated monkeys and died on December 25 and 27, with typical and complete paralysis. The periods of incubation, the course of the disease, the severity of the infections, and the autopsy findings differed in no essential way from those of the immunized monkeys.

Monkey No. 108 was immunized against typhoid by repeated inoculations of an antityphoid vaccine. December 3, 400,000,000 were given intraperitoneally. December 8, 400,000,000 were given intraperitoneally. December 15, 800,000,000 were given intraperitoneally. After an interval of ten days, to allow any immunity that might be produced to be at its height, on December 26 1 cubic centimeter of cord M. A. emulsion was given intracranially: —

1912.
January 1, a little nervous, but no paralysis.
January 2, beginning paralysis.
January 3, complete paralysis.
January 6, death with typical autopsy findings.

Monkey No. 109 received three inoculations with an anti-gonococcus vaccine intraperitoneally as follows: —

1911.	
December 3,	100,000,000
December 8,	100,000,000
December 15,	100,000,000

On December 26, after allowing ten days for any immunity to appear, 1 cubic centimeter of the M. A. cord emulsion was given intracranially: —

1911.
December 30, beginning paralysis.
December 31, complete paralysis.

1912.
January 1, died with typical autopsy findings.

Monkey No. 110 was given three injections with a pertussis vaccine, obtained from the Parke, Davis experimental laboratories, as follows: —

1911.

December 3,	20,000,000
December 8,	40,000,000
December 15,	40,000,000

On December 26 received 1 cubic centimeter emulsion from M. A. cord intracranially: —

1911.

December 30, first signs of paralysis.

1912.

January 1, complete paralysis and death, with typical autopsy findings.

Monkey No. 111 received three injections with a staphylococcus pyogenes aureus vaccine intraperitoneally as follows: —

1911.

December 3,	500,000,000
December 8,	1,000,000,000
December 15,	1,000,000,000

December 30, first signs of paralysis.

1912.

January 12, complete paralysis and death, with typical autopsy findings.

Nos. 119 and 120, control monkeys, were inoculated December 26 with 1 cubic centimeter emulsion cord, M. A. Both showed the beginning of paralysis on the 30th. January 1, 1912, complete paralysis, death, and typical autopsy findings. The periods of incubation, the course of the disease, and the severity of the infections corresponded closely to those of the immunized monkeys.

The inoculations of the virus of poliomyelitis were given at a time when the supposed artificially produced immunity was strongest, and although the virus was active and doses of considerable size were used, no protective phenomena attributable to the immunized injections were noted in any instance, with the possible exception of the monkey immunized against typhoid, which was a little slower in showing signs of paralysis and lived a little longer after the onset than the controls. Such a slight variation, however, it seems to us, may well be attributed to the increased natural resistance of this individual monkey to poliomyelitis or the strength of the inoculated emulsion. It is interesting to note that at autopsy marked tubercular lesions were present in this monkey (No. 103) as well as those of poliomyelitis.

These few experiments seem to show, therefore, that in the *Rhœsus* monkey a partial and perhaps a complete immunity to the infection of dysentery, streptococcus pyogenes, typhoid fever, gonorrhœa, pertussis (whooping cough), and staphylococcus pyogenes affords no demonstrable protection against the virus of poliomyelitis.

That these conclusions are justified has received recently confirmation from the results of Landsteiner and Levaditi (Annales de l'Institut Pasteur, November, 1911), who found that specific inoculations carried out with the virus of rabies, a disease much more closely allied to anterior poliomyelitis than any of the infections above detailed, produced in monkeys no immunizing effect whatever. Furthermore, the reverse was true, that inoculations with anterior poliomyelitis virus produced no protective effects against rabies.

An idea of the detail and scope of these studies may be had from the appended chart.

Monkey No.	Immunized against —	Date and Amount of Immunizing Injections.	Date and Amount of Inoculation with Virus of Poliomyelitis.	Date of Onset of Paralysis.	Date of Death.	Autopsy Report.
106	Dysentery, . .	Dec. 3, 1911, 1 cubic centimeter anti-dysenteric serum intraperitoneally. Dec. 8, 1911, same dose. Dec. 15, 1911, same dose.	Dec. 18, 1911, 2 cubic centimeters emulsion, intracerebrally.	Dec. 22, 1911	Dec. 24, 1911	Meninges and cord congested. Lumbar cord, typical lesions of poliomyelitis.
107	Streptococcus, .	Dec. 3, 1911, 10 cubic centimeters anti-streptococcus serum intraperitoneally. Dec. 8, 1911, same dose. Dec. 15, 1911, same dose.	Dec. 18, 1911, 2 cubic centimeters emulsion, intracerebrally.	Dec. 22, 1911	Dec. 26, 1911	Sections of cord show typical lesions of poliomyelitis.
117	Nothing (Control Monkey).	– –	Dec. 18, 1911, 2 cubic centimeters emulsion, intracerebrally.	Dec. 23, 1911	Dec. 25, 1911	Sections of cord show typical changes of poliomyelitis. Anterior horns swollen.
118	Nothing (Control Monkey).	– –	Dec. 18, 1911, 2 cubic centimeters emulsion, intracerebrally.	Dec. 23, 1911	Dec. 27, 1911	Cord and brain congested. Characteristic lesions of poliomyelitis.
108	Typhoid, . .	Dec. 3, 1911, 1 cubic centimeter anti-typhoid vaccine, intraperitoneally, 400,000,000. Dec. 8, 1911, same dose. Dec. 15, 1911, 2 cubic centimeters of same vaccine, 800,000,000.	Dec. 26, 1911, 1 cubic centimeter emulsion, intracerebrally.	Jan. 3, 1912	Jan. 6, 1912	Marked signs of tuberculosis in spleen, lungs and mesenteric glands not the cause of death. Cord shows typical lesions of poliomyelitis.
109	Gonococcus, .	Dec. 3, 1911, 1 cubic centimeter anti-gonococcus vaccine, intraperitoneally, 100,000,000. Dec. 8, 1911, same dose. Dec. 15, 1911, same dose.	Dec. 26, 1911, 1 cubic centimeter emulsion, intracerebrally.	Dec. 31, 1911	Jan. 3, 1912	Meningeal vessels congested over cord and brain. Cross-section of cord shows no marked change in horns macroscopically. Microscopically typical changes of poliomyelitis with atypical changes of degeneration in some of the nerve cells.
110	Pertussis, . .	Dec. 3, 1911, 1 cubic centimeter anti-pertussis vaccine, intraperitoneally, 20,000,000. Dec. 8, 1911, 2 cubic centimeters. Dec. 15, 1911, 2 cubic centimeters.	Dec. 26, 1911, 1 cubic centimeter emulsion, intracerebrally.	Dec. 31, 1911	Jan. 3, 1912	Vessels, cord and brain congested. Anterior horns swollen. Cord shows typical changes of poliomyelitis.

Monkey No.	Immunized against —	Date and Amount of Immunising Injections.	Date and Amount of Inoculation with Virus of Poliomyelitis.	Date of Onset of Paralysis.	Date of Death.	Autopsy Report.
111	Staphylococcus, .	Dec. 3, 1911, 1 cubic centimeter anti-staphylococcus vaccine, intraperitoneally, 500,000,000. Dec. 8, 1911, same dose. Dec. 15, 1911, 2 cubic centimeters.	Dec. 26, 1911, 2 cubic centimeters emulsion, intracerebrally.	Dec. 31, 1911	Jan. 2, 1912	Vessels of cord and brain slightly congested. Anterior horns not swollen. Cord shows typical changes of poliomyelitis. Some changes in nerve cells (neurophagia) that are unusual.
119	Nothing (Control Monkey).	– –	Dec. 26, 1911, 1 cubic centimeter emulsion, intracerebrally.	Dec. 31, 1911	Jan. 2, 1912	Sections of cord show typical lesions of poliomyelitis.
120	Nothing (Control Monkey).	– –	Dec. 26, 1911, 1 cubic centimeter emulsion, intracerebrally.	Dec. 31, 1911	Jan. 2, 1912	Vessels of brain and cord congested. Infiltration of cells about anterior horns which suggests poliomyelitis. Nerve cell degeneration, neurophagia, etc., unusual. Probably atypical poliomyelitis.

PROGNOSIS IN INFANTILE PARALYSIS.[1]

By B. E. Wood, M.D., Boston, Mass.

At the request of the Massachusetts State Board of Health, in April, 1911, I undertook the investigation as to the present condition of those cases of infantile paralysis which were reported to the Board in the year 1907, in order to ascertain the number of recoveries occurring in the four years and thereby to increase our knowledge concerning the prognosis in this disease.

Letters were sent to all physicians who had reported cases in 1907, asking whether or not such cases had recovered and, when recovery was reported, such cases were seen personally unless they had disappeared or moved. If they could not be seen, all available information was obtained about them.

Of the 234 cases, 22 had disappeared, leaving 212 the termination of which is known; of these, 57 (27 per cent.) were reported as having recovered; 11 died in the acute stage of the disease and 8 others subsequently died of other diseases, 2 of these having recovered from the paralysis.

It was found that there were three classes of recovery: (1) Complete recovery without atrophy, (2) recovery with complete function but with some atrophy, (3) recovery with some hypertrophy of the affected limb.

The table shows the proportion of each of these:

		Per Cent.
Complete recovery without atrophy,	16	28.1
Functional recovery with atrophy,	21	36.8
Recovery with some hypertrophy,	3	5.3
Recovery, presence or absence of atrophy unknown,	17	29.8

Leaving out the last group, and averaging those in which the presence or absence of atrophy is known, shows that for every 4 recoveries without atrophy there were 5 with atrophy. When atrophy was present the maximum amount noted was as follows: —

	Inches.
Calf,	1¾
Thigh,	1¼
Arm,	½
Forearm,	¾

[1] Reprinted from Boston Medical and Surgical Journal, Oct. 5, 1911.

In most cases the atrophy was much less than this, a difference of one-eighth of an inch being counted as atrophy, but in all such cases examined function of every muscle was perfect. Three cases had only one-eighth inch atrophy of one limb; 2 cases only one-fourth inch of one limb; 5 cases only one-eighth inch of one limb and one-fourth inch of another; the other 11 cases had more than one-fourth inch atrophy. The amounts of hypertrophy recorded were in two instances one-fourth inch and in one three-eighths inch, all of the calf. One of these cases recovered in two months and has had intermittent massage up to date; one recovered in three weeks, having had daily massage for that time; one recovered in three months and had daily massage for six weeks. The duration of the treatment apparently does not account for the hypertrophy in the last two cases.

The severity of the attack in the recovered cases was classed as: —

		Per Cent.
Severe,	14	24.5
Moderate,	9	15.9
Mild,	28	49.1
Not noted,	6	
Total,	57	

The distribution of the paralysis was as follows: —

One lower extremity,	12
Both lower extremities,	9
One upper extremity,	6
Both upper extremities,	2
One lower and one upper extremity,	10
Three extremities,	7
Four extremities,	5
Face alone,	1
General,	1
Not given,	4
Total,	57

In addition to this distribution, paralysis of the face also existed in four, of the back in five, of the face and back in one, and of the abdominal muscles in one.

The time of recovery in the 57 cases was as follows: —

1 week or less,	2
1 week to 1 month,	8
1 to 2 months,	8
2 to 3 months,	5
3 months to 6 months,	10
6 months to 12 months,	9
1 to 2 years,	5
2 to 3 years,	5
No data,	5
Total,	57

To illustrate late recovery, and recovery from severe attack, two cases were especially notable.

W. H. and J. H., brothers, age eleven and thirteen, patients of Dr. J. C. Hubbard, of Holyoke, were attacked Oct. 12 and 13, 1907, respectively.

The duration of the acute attack was three weeks in each case, that of W. H. being moderate and that of J. H. severe. The latter had a temperature of 103° F. for a week, vomiting for several days and severe coma for two weeks, with marked retraction of head. The distribution of his paralysis at its worst was both thighs, left leg, left arm and left forearm and lower back. The arm and back recovered in one month.

The distribution of W. H.'s paralysis was left thigh and left leg.

The treatment was the same in both cases and consisted of massage and tabetic exercises begun in November, 1907, after the acute onset.

Both patients were in bed three weeks and had to be carried for two months and then walked with a limp, the lameness being very marked in the case of J. H. during the first year. Massage was given every night for two years. The greatest gain occurred during the second year and practical recovery occurred during the third year.

An examination in May, 1911, showed no paralysis in either case. W. H. had ⅛ inch atrophy of left calf and ½ inch of left thigh. J. H. had ⅛ inch atrophy of right calf and no atrophy of the thigh or arm.

Very little can be said as to the effect of treatment. Massage, carried out more or less irregularly in most cases, was the most common procedure, being used in 36 of the recovered cases and alone in 25, with exercises in 5, and with baths, hot and cold, in 4. Electricity was used alone in 1 case, and with massage in 5 cases. Drugs alone were used in 12 cases, the most commonly used being strychnia and K. I. There was no treatment in 5 cases. The cases in which there was no treatment, or in which drugs alone were used, were, as a rule, those which recovered early and which did not, therefore, need other treatment.

The following conclusions seem justified: —

In anterior poliomyelitis complete recovery or functional recovery occurs in something over 25 per cent. of cases examined at the end of four years. Atrophy may exist without impairment of function. In about half of the recovered cases, the onset was mild. The distribution of the paralysis in such recovered cases was not essentially different from that in cases which do not recover. Recovery in many instances required months, and in several cases from one to three years.